THE JOURNAL OF PERIODONTOLOGY

January
1931

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME II.
NUMBER I.

THE Journal of Periodontology is lovingly dedicated to the memory of Doctor Gillette Hayden from whose achievements in the preparation of dental educational bulletins it is an outgrowth. Her selfless devotion and untiring efforts in behalf of the science of periodontology, the art of periodontia and the American Academy of Periodontology, have served as an inspiration to her close associates which can only be consummated by carrying onward the work for which she spent her life.

Preface

THE SECOND number of Volume I of the Journal of Periodontology was not published as planned owing to the delay in receiving the abstracts which were to have made up the greater portion of that issue. The tremendous task which the committee had assumed in attempting to cover so much territory in the line of abstracting readily explains the delay. An effort was made to abstract all the articles bearing on periodontology which appeared during 1927—'28 and '29, and since there were approximately six hundred it was impracticable to complete them in so short a time. The Committee on Journal Publication trusts that the value of the work when completed will more than offset any disappointment caused by delay in its publication.

Since Academy members often are residing in parts of the United States and Canada at a great distance from the place of holding the annual meeting, it has been thought best to publish a few reports of committees and other information, which although without especial interest to those outside the Academy membership, is at least a means of putting members unable to attend meetings regularly in touch with the activities of the organization.

This number of the Journal contains in addition to the above mentioned material the program for the next annual meeting in Memphis, Tennessee; a bibliography of important literature bearing on periodontology; abstracts of articles published during a three year period on Peridontal Histopathology, Periodontal Bacteriology, and Other Laboratory Research (dietetics, physiological chemistry, etc.). Abstracts on Etiology from a Clinical Standpoint; Symptoms and Diagnosis; Treatments; Home Care and Prophylaxis; Vincent's Gingivitis; Miscellaneous; will follow in a later issue. It is recommended by the committee that each member of the Academy place these numbers of the Journal on file since they will be invaluable for reference and will no doubt be much in demand by students, writers, schools and libraries.

The reader's attention is called also to the excellent program arranged for the next annual meeting in October. No one interested in periodontia should miss it since it is of a high order most carefully thought out in every detail. The program committee and the Academy are to be congratulated.

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THIS CHART WILL BE THE FIRST CHART
APPEARING AT THE END OF THIS FILM

Announcement

THE EIGHTEENTH ANNUAL meeting of the American Academy of Periodontology is to be held at the Hotel Gayoso, Memphis, Tennessee, Thursday, Friday, and Saturday, October 15-16-17, 1931.

The Hotel Gayoso is convenient and is in a position to provide ample facilities for the Scientific, Clinical, and Entertainment programs.

Members and guests should make their reservations now to insure accommodations.

CLAYTON H. GRACEY,
Secretary.

966 Fisher Building,
Detroit, Mich.

The Meeting At Colorado Springs

It was an enthusiastic group of periodontists that gathered at the Acacia Hotel in Colorado Springs in July, 1930 to enjoy an excellent three day program and to experience true western hospitality. The members in Colorado together with the efficient officers and committees of last year had anticipated every wish and of approximately one hundred who had registered, all left their western friends, the comfortable hotel, and the beautiful city with regret but with a broader vision, higher aspirations, and increased knowledge of periodontia.

Reports of Committees

Committee on Journal Publication

AT THE MEETING last year this committee was authorized to undertake a departure from the work of previous years. Instead of preparing more bulletins for the laity or dentists it has launched a Journal of Periodontology the first number of which was issued as of January 1930, and was sent to every member of the Academy in March.

There are several reasons for having the Academy issue a Journal of Periodontology. There is a very great need in all dentistry for an abstracting bureau since the literature is getting so large. It is particularly appropriate that this Academy should attend to the abstracting of the literature dealing with periodontology: the Journal can be the means of doing this. In the second place if the field of periodontology is to receive its maximum scientific development it should have its own journal for original articles just as the Journal of Bacteriology or the Journal of Biological Chemistry serve as the first publishing place for articles in these fields. If any subject is to develop scientifically it must develop its own literature. In the third place this seems a fitting method of building a memorial to our beloved Gillette Hayden whose ideals for the Academy lay along the lines of productive labor.

The second issue of the Journal will contain no original articles but will be devoted to a complete bibliography of all important literature bearing on periodontology, and abstracts of all such literature from December 1926 to January 1930, together with reviews of all books dealing with the subject.

The Council has authorized the Committee to proceed with the second years volume and it is the intention to begin in this the publication of the best of the original articles in our field. Since there will certainly be a demand for the Journal from dental schools, libraries and many dentists, the Committee expects to have 750 copies of each number printed and offered for sale at the cost of issuance. It is planned that they be advertised and announced in the leading dental journals. The January number each year will contain the minutes, proceedings, notices, etc. of the Academy.

The Academy of Periodontology should be the great producing center of knowledge, research, and publication in the field of periodontology. It is hoped that the preparation and publication of this journal may serve as a foundation for expanded activity in these fields.

Respectfully submitted,

ARTHUR H. MERRITT
CHARLES B. FOWLKES
M. M. BETTMAN
CELIA RICH
MELVILLE QUINBY
GRACE ROGERS SPALDING
HAROLD J. LEONARD, *Chairman*

Committee on Scientific Investigation

The Committee on Scientific Investigation presents a report by Dr. Hirschfeld on "The Diagrammatic Recording of Periodontal Diseases." This is an elaboration of a brief talk which was given by him at the meeting of the International Society for Dental Research in Toronto, last March and will be illustrated by charts and photographs. The presentation of this subject should prove interesting because it represents the first step in the proper conduct of cases; an accurate record means much to a periodontist. Dentists, as a group, have not given the practise of recording cases the thought and attention that it merits and as a consequence, the average dental case history

loses much value when compared with one made by the physician in his practise. Dr. Hirschfeld's presentation should furnish us with some new ideas of scientific value.

Members, who are interested in original problems in periodontology, which may or may not be thoroughly established, are once more requested to cooperate with this committee by contributing reports on the subjects whenever sufficient material is gathered. Work of this kind need not necessarily concern laboratory findings because such research is not possible or available for the average practitioner. Problems in clinical periodontia are just as important as those of the laboratory and contribute as valuable information to the profession at large, because in the end, it is the practical application of research findings of all kinds that gives the greatest benefit to the patient; laboratory reports are but a means to an end.

While it is evident that the International Society of Dental Research has begun an important undertaking in the interests of dentistry in general, it seems that the first obligation of members of the Academy should be to submit original material to the Committee on Scientific Investigation for presentation at the meetings and thus encourage the efforts of our organization to promote new knowledge in periodontology.

A recommendation is hereby made that the Secretary be instructed to mail a copy of this report to every member of the Academy hoping thereby to stimulate interest in research.

Respectfully submitted,

THOMAS B. HARTZELL,
JOHN OPPIE MCCALL,
I. HIRSCHFELD,
ARTHUR H. MERRITT.
BENJAMIN TISHLER, *Chairman*

1 1 1

Committee on Bulletin Distribution

The report made to the Council in January stated that in order to give a wide distribution to the newspaper publicity articles, it was planned to send a copy to all members of the Academy asking them to pay an amount which would cover the cost of printing and distribution.

Said report also stated that the approximate cost of printing would be about two hundred dollars and the committee asked the Academy to underwrite that amount with something additional for postage. Owing to a misunderstanding in the printing contract, the articles were set up in small book form, which did not seem suitable for newspaper work, so at an added cost of a small amount five hundred sets were printed in a suitable form for newspapers. When the final printing had been completed, the cost of printing for the five hundred newspaper sets, one thousand small booklets, special envelopes and five hundred form letters amounted to $328.75. By making a charge of two dollars for each newspaper set and fifty cents for each booklet, the committee believed the cost of distribution could be met.

In March, 1930, the articles were ready for distribution. A copy was sent to each member of the Academy asking that member to aid in the distribution by purchasing one set and giving it to a newspaper in his locality for publication. In the short time which has elapsed, we feel that considerable has been accomplished. One hundred and nineteen have responded with checks and only six sets have been returned. On June 15th a follow-up letter was sent to all members from whom we had not heard. The response has been most gratifying. Many members have stated that they were unable to get them in their newspapers, but were glad to own a set for themselves and some members have bought extra copies.

This committee is glad to report to the members of the committee who wrote and compiled the articles that every one who has received them has made favorable comment on the material.

At this time it is impossible to give a

definite report as to the number of states in which they are being published. Clippings of these articles have been received from the following newspapers: Aberdeen American-News, Aberdeen, S. D.; The Gazette & Telegraph, Colorado Springs, Colo.

From correspondence, it is learned that the following will publish some of the articles: The Journal, Portland, Oregon; Buffalo Evening News, Buffalo, N. Y.; Bedford Co. Times, Shelbyville, Tenn.; The Mississippi Guide, Gulfport, Miss.; Fayette Co. Dental Society, Uniontown, Pa.

Wisconsin State Dental Society is sponsoring them in newspapers throughout the State of Wisconsin.

Harrisburg, Pa., Dental Society is using them for broadcasting.

N. Y. Tuberculosis & Health Association are using some in their health education program.

Publication is also being considered by the following: Western Fairfield County Dental Society, Conn., Kansas City Star, First District of Michigan, One of the Chicago Dental Study Clubs, Baltimore, Md., Oral Hygiene Com., Michigan Dental Society, Holton, Kansas, Manistee County Medical Society, Manistee, Mich., Miami, Fla., Macon, Ga., Hartford, Conn., Columbus Despatch, Columbus, Ohio.

The American Dental Journal published a notice in the June issue, page 1156, relative to these articles and we have had a number of requests turned over to us by the Journal recently. One order has come from Berlin. In addition to the form letters sent out with the sets of articles, there were more than one hundred and fifty personal letters written to members of the profession most of whom were non-members of the Academy. Your chairman while in attendance at the Southern Academy of Periodontology in Atlanta last March disposed of 18 sets, reaching districts not represented by the American Academy.

At the present time, we are negotiating with the Western Newspaper Union which supplies articles for four thousand small town papers; while we have no assurance that they will be accepted, the committee desires expression from the Academy relative to such an arrangement.

As per the attached financial report, all of the expenses of printing and postage have been paid leaving a balance of cash on hand of $24.54. Also have on hand 120 sets of the newspaper forms and 900 of the booklets; the latter the committee suggests be put in circulation after the articles have been run in the public print so that they will in no wise interfere with the newspaper publicity; these books can also be used to great advantage in school and mouth hygiene work and also private practice.

We trust that the members of the Academy who have been so willing to aid in this work financially will feel that their money has been well spent in the manner of distribution. There is a great need for such publicity as this but it requires the interest of every member to promote it.

Respectfully submitted,

SIDNEY RAUH,
EDWARD L. BALL.
CARLOS H. SCHOTT, *Chairman,*

Round Table Discussions

IT HAS BEEN a custom to hold Round Table Discussions during the luncheon hour on one day at the annual meetings of the Academy, topics for discussion and leaders being chosen in advance and announced in the regular program. This plan has been a popular one since it affords all those present an opportunity for free interchange of ideas on the subjects under discussion and gives the added advantage of a better understanding of the point of view of fellow practitioners.

There have been many requests to have the reports of the Round Table Discussions published. If it be kept in mind that these reports do not necessarily represent the opinions of the majority in the Academy membership but simply express the conclusions arrived at by those few who happened to discuss the particular subject assigned, they will be of interest.

July 18th, 1930.

DR. C. W. HOFFER, *Chairman*

Table No. 1. What factors govern a favorable prognosis in periodontal treatment?

DR. A. C. HAMM, *Leader*

REPORT—

1. Normal physiological occlusion.
2. Distribution of bone destruction.
3. Co-operation and health habits of the patient.
4. General health of the patient.
5. Resistance, or immunity of the patient to disease.

Table No. 2. What type of periodontal disease is most amenable to treatment?

DR. F. A. BRICKER, *Leader*

REPORT—

The type having hypertrophied tissues, gross deposits and showing great neglect.

Table No. 3. Is there any relationship between the etiology of periodontoclasia and cervical erosion?

DR. M. M. BETTMAN, *Leader*

REPORT—

A radical difference of opinion due to a lack of agreement concerning the etiology of erosion.

Table No. 4. Do you consider traumatic occlusion to be an important etiological factor of periodontoclasia?

DR. C. S. KRAMER, *Leader*

REPORT—

Generally *no,* usually a result of periodontoclasia. However due to uneven wear, inlays, bridges and fillings which are out of occlusal harmony may possibly be the cause of local irritation, the stress of which the individual structure is unable to bear, hence becoming in local areas important etiological factors in periodontoclasia.

Table No. 5. Can traumatic occlusion be corrected in all mal-occlusion cases by grinding? State exceptions, if any.

DR. PAUL A. EDMAND, *Leader*

REPORT—

No. The exceptions: excessive overbite; excessive open-bite; where the median line has shifted over the width of one tooth; irregular cases such as torso-occlusion; cases influenced by an over-developed third molar. It was agreed that some grinding might help these cases but would not entirely correct them.

Table No. 6. Do the gingivae of all patients respond alike to tooth brush massage? If not, why? DR. L. A. NEIL, *Leader*

REPORT—

They do not. Among the causes for this are: improper function; health habits of the patient; physical debilities; nutritional unbalance.

Table No. 7. What factors prolong the treatment period of Vincent's infection?

DR. JUSTIN D. TOWNER, *Leader*

REPORT—

Smoking; excessive use of alcoholics; poor nutrition; lack of co-operation; neglect of the case due to tardy diagnosis; neglect by the patient to seek early relief; lack of mouth hygiene; periodontal pockets where organisms can grow undisturbed; traumatic occlusion. (At this table were seated dentists from California, Tennessee, Florida, Washington State, Ohio, Iowa, Texas and Honolulu.)

Table No. 8. Do you consider that there is a relationship between the horizontal and

vertical types of periodontoclasia and Vincent's infection?

DR. OLIN KIRKLAND, *Leader*

REPORT—

(No general conclusion reached.) Individual opinions: "There is a relationship between both forms and Vincent's Infection in that anything that lowers tissue tone, superinduces the latter disease." "Seldom if ever is Vincent's Infection found in a mouth not showing signs of periodontoclasia." "Vincent's Infection plays an important part in ninety per cent of all periodontoclasia."

* * *

Gillette Hayden Memorial

A dental department in the Sarah Hackett Stevenson Home, Chicago, was dedicated to the memory of the late Dr. Gillette Hayden, on February 3rd, 1931. The Memorial is to be devoted to the service of children and is in every way a fitting tribute to her memory.

The following poem by Florence Holbrook read at the dedication reflects the thought of Doctor Hayden's many friends:

* * *

G ifted in all the arts of life, our friend,
I nspirer in us of all noble aims,
L ived every day the seven glowing flames,
L ove, joy, and work, and patience without end,
E nduring calm, humility, and grace,
T rained in the schools she gave her generous heart
T o rousing other minds to do their part,
E ager to advance the progress of the race.

H ow well she has attained her earthly goal
A ll our profession loyally attest,
Y et other heights called to her noble soul—
D auntless she faced the unknown Future's quest,
E ntered the realm of everlasting light,
N e'er lost to us—just gone beyond our sight.

—*Florence Holbrook.*

Brief Report of the Mid-winter Council Meeting

JANUARY 31, 1931, CHICAGO

THE MEETING was called to order at 10:00 o'clock, by Dr. Carl W. Hoffer, in the absence of the Chairman, Dr. Clyde C. Sherwood.

The following members of the Council were in attendance: Arthur H. Merritt, Grace Rogers Spalding, Benjamin Tishler, Carl W. Hoffer, Harold J. Leonard, W. H. Scherer, J. C. Salvas, Clayton H. Gracey.

The Secretary's minutes were read and approved.

The report of the Treasurer in the form of an audit by the company, Mounteer, Steveling and Wyllie, was submitted and accepted.

The following Committee reports were submitted and accepted:

Program, Clinics, Scientific Investigation, Publication and Journal Publication.

A Committee on Credentials was appointed, composed of the following members: Roy O. Elam, Robert L. Dement, Clayton H. Gracey.

A motion was carried to the effect that the Newspaper Bulletins, compiled by the A. A. of P. may be distributed by any recognized and authorized organization, under that organization's name, which shall in turn, give credit to the A. A. of P. for their compilation.

A motion was carried that the use of a reporting agency be discontinued at our next meeting.

A Committee of two was appointed to submit a report on a plan for a division of duties of the Secretary.

J. D. Towner was appointed to fill the unexpired term on the Council of Harold J. Leonard, to 1932, the latter having been made President-elect at the last annual meeting.

The Secretary was instructed to send a registered letter to delinquents, requesting an early payment of dues, and inclosing a quotation from the Constitution, referring to delinquents.

Many other topics of minor but nevertheless vital importance to the welfare of the Academy were thoroughly discussed, resulting as usual in a valuable mid-winter council meeting.

Important Notice

All members of the American Dental Association are cordially invited to attend the Academy meeting in Memphis, Oct. 15th, 16th, and 17th, 1931, at the Hotel Gayoso.

Memorial Resolutions, 1930

WHEREAS, There has been taken from our midst since last we met together in annual session, three of our most honored and respected members:

John Dean Patterson, D. D. S., F. A. A. P.
Frank H. Skinner, D. D. S.
Frank C. Pague, D. D. S. and

"WHEREAS, It is with a profound sense of loss that we take note of their passing and avail ourselves of this opportunity to record our esteem for them and pay reverence to their memories, and

"WHEREAS, We shall miss their presence among us and with a deep sense of loss record the kindliness and wisdom of their counsel which was always so fully given in the interest of their colleagues and for the upbuilding of their profession to which they gave themselves with such unselfish devotion; therefore, be it

"RESOLVED, That we pay tribute to their sterling qualities and with bowed heads and chastened spirits pledge ourselves to uphold the high ideals which characterized them in all their relations with their fellow men; that these resolutions be made a part of the permanent records of this Academy, and that a copy be sent to the members of their families and to the journals for publication.

AUSTIN F. JAMES,
ARTHUR H. MERRITT,
JUSTIN D. TOWNER, *Chairman.*

In Memoriam
1931

DR. LITA SHRINER
DR. JULIAN SMITH
DR. RICHARD C. WALDEN

Program for the Eighteenth Annual Meeting

HOTEL GAYOSO, MEMPHIS, TENNESSEE

THURSDAY, OCTOBER 15, 1931

9:00 A.M.	Registration	
9:30 A.M.	Opening of Eighteenth Annual Session	DR. CARL W. HOFFER, *President*
	Report of Secretary and Treasurer	C. H. GRACEY
	Report of Program Committee	A. H. MERRITT
	Report of Clinic Committee	BENJAMIN TISHLER
	Report of Bulletin Distribution Committee	C. H. SCHOTT
	Report of Journal Committee	H. J. LEONARD
	Report of Nomenclature Committee	E. M. QUINBY
	Report of Publication Committee	GRACE R. SPALDING
	Report of Arrangement Committee	C. H. GRACEY
11:00 A.M.	President's Address	
11:30 A.M.	Report of the Eighth International Dental Congress	
2:00 P.M.	"Observations of Clinical Manifestations of Fuso-Spirochetosis"	RICHARD D. DEAN, B. S., D. D. S., M. D.
3:00 P.M.	"Observations of Cultural Characteristics of Fuso-Spirochetes"	M. T. DEAN, B. S., M. D.
4:00 P.M.	Incidents of Office Practice (Papers limited to ten minutes)	
	"Loss of Teeth Especially the Mandibular First Molars, as a Factor in Periodontoclasia"	DR. ARTHUR C. HEIBERT
	"The Patient Whom you see Every Three or Four Years"	DR. WALTER LEABO
	"Everyday Questions and Answers in Reference to Vincent's Infection"	DR. SAUL L. SCOTT
	"Periodontal Diagnosis by Biopsy; a Case Report"	DR. HARRY LYONS
	"Retraction and Maintenance in Normal Position of a First Superior Incisor"	DR. REUBEN LENZER
	"The Importance of Early Diagnosis"	DR. ROBERT L. DEMENT
	"Some Observations on the Effects of Trauma"	DR. S. PRESTON
8:00 P.M.	"Biological Aspects of Dental Practice"	DR. F. V. SIMONTON

FRIDAY, OCTOBER 16

9:00 A.M.	Report of the Committee on Scientific Investigation	BENJAMIN TISHLER, *Chairman*
	"Researches in Periodontology Going Forward at Columbia University"	DR. HAROLD J. LEONARD
	"Hairy Tongue (Lingua Negri) As a Sequence to Vincent's Infection Treatment; A Report of Three Cases" (Illustrated by lantern slides)	DR. ISADOR HIRSCHFELD

"The Tongue in its Relation to Periodontal Disease" DR. T. B. HARTZELL

"Recent Advances in the Field of Periodontology" (Illustrated by lantern slides) DR. BERNARD GOTTLIEB

(There will be a question period after each contribution if time will permit.)

12:00 M. Round Table Discussion DR. R. HOLMES MASON, *Chairman*

1. In what way could the programs of the Academy be improved. DR. CARLOS H. SCHOTT, *Leader*
2. In what way can the Academy aid in promoting greater interest in periodontia. DR. I. HIRSCHFELD, *Leader*
3. How should the dentist be educated to best meet the needs of the public. DR. JOHN OPPIE MCCALL, *Leader*
4. How can post-graduate study in periodontia be made most effective. DR. JUSTIN D. TOWNER, *Leader*
5. What should be the scope of periodontia DR. HAROLD J. LEONARD, *Leader*
6. Does the present day education of the dentist qualify him to give dietetic advice to his patients? DR. GRACE R. SPALDING, *Leader*
7. How should patients be taught to care for their mouths? DR. OLIN KIRKLAND, *Leader*
8. Should the dental hygienist be intrusted with the post-operative care of patients who have been treated for periodontoclasia? DR. E. MELVILLE QUINBY, *Leader*

2:30 P.M. "The Value of Blood Examination in the Practice of Periodontia" IRWIN A. EPSTEIN, D. D. S.

3:30 P.M. Topic Discussion DR. CLYDE C. SHERWOOD, *Chairman*
DRS. OLIN KIRKLAND AND ARTHUR H. MERRITT, *Leaders*

1. Of what should periodontia consist?
2. What are the types and differential diagnosis in Vincent's infection?
3. What principles govern its treatment?
4. What are the factors favoring the deposition of salivary and serumal calculus?
5. What are the etiologic factors in periodontoclasia? How may they be classified and simplified?
6. Define traumatic occlusion and its influence in periodontal diseases.
7. Can one distinguish between periodontoclasia of systemic or local origin?
8. What are the different types of operations for the treatment of periodontoclasia, and what are the advantages and disadvantages of each?
9. Why do you favor one method of treatment over another?
10. In cases in which the patient has been referred by a dentist or physician, what are the obligations of the periodontist?
11. Of what does the post-operative care of the patient consist?
12. Who should be held responsible for this, the family dentist or the periodontist?
13. What are the possibilities and limitations of prevention?
14. What are the economic problems of periodontia and how may they be solved?

8:00 P.M. "Individual Occlusal Requirements in its Relation to Investing Structure and Teeth." DR. HARRY G. MORTON, Milwaukee, Wis.

SATURDAY, OCTOBER 17

9:30-10:30 A.M. General Clinics

Agfa Color Slides. Selected Cases Before and After Treatment: Subgingival Currettage — DR. C. B. FOWLKES, New York

Restoring Tooth Form in the Correction of Traumatic Occlusion — DR. W. H. SCHERER, Houston, Texas

Vincent's Infection of the Mouth (Illustrated by photographs) — DR. ISADOR HIRSCHFELD, New York

Restoring Mutilated Mouths — DR. ROY O. ELAM, Nashville, Tenn.

Oral Manifestations of Systemic Diseases — DR. WILHELMINA YERETSKY, Grand Rapids, Mich.

Practical Operating Methods in Periodontal Lesions — DR. WALTER LEABO, Shreveport, La.

(Illustrations of interesting cases as shown by lantern slides)

10:35-11:35 A.M. A Clinical Demonstration of the Conservative and Radical Methods of Root Currettment.

Conservative Method

DR. AUSTIN F. JAMES, Chicago, Ill.

DR. THOMAS F. BATES, Shelbyville, Tenn.

Radical Method

DR. OLIN KIRKLAND, Montgomery, Ala.

DR. ROBERT L. DEMENT, Atlanta, Ga.

(Patients will be furnished to these clinicians)

11:40 A.M.-12:40 P.M.

Questionnaire on Periodontia

DR. PAUL R. STILLMAN, New York, Consultant

7:00 P.M.....*Annual Banquet*

BIBLIOGRAPHY, REFERENCES AND ABSTRACTS RELATING TO PERIODONTIA

CONTENTS

Foreword 21

Bibliography of Periodontia and References 23

Abstracts and Extracts of Current Literature for 1927-'28-'29:

(*a*) Periodontal Histopathology 35

(*b*) Periodontal Bacteriology 75

(*c*) Other Laboratory Researches (dietetics, physiological chemistry, etc.), Related to Periodontology 84

Compiled and Published by

The American Academy of Periodontology

in the

JOURNAL OF PERIODONTOLOGY

JANUARY, 1931

FOREWORD

This number marks the beginning of an attempt to bring together the scattered literature bearing on periodontology in a form available to students and practitioners. An effort has been made to list all books bearing on the subject and to assemble all the current literature as abstracts, starting with January, 1927.

The articles are catalogued under the headings:

(a)—HISTOPATHOLOGY
(b)—BACTERIOLOGY
(c)—OTHER LABORATORY RESEARCH
(d)—ETIOLOGY FROM A CLINICAL STANDPOINT
(e)—SYMPTOMS AND DIAGNOSIS
(f)—TREATMENTS
(g)—HOME CARE AND PROPHYLAXIS
(h)—VINCENT'S GINGIVITIS
(i)—MISCELLANEOUS

Abstracts for the years 1927, 1928, and 1929 on the first three subjects are included in the present number; those for the remaining subjects of these three years are now being prepared and will appear in the next issue. Later numbers will contain the abstracts for 1930 and succeeding years.

The work is being done under the auspices of the Committee on Journal Publication of the American Academy of Periodontology. The titles of the articles are assembled in the office of the Chairman and distributed for abstraction to the members of the Committee and others of the Academy who have volunteered. Suitable abstracts appearing in other journals have been copied or translated with due acknowledgment. The present series has been abstracted by the following persons, the initials of the writer appearing after each abstract: Arthur H. Merritt, D. D. S., of New York City; Celia Rich, D. D. S., of Nashville, Tenn.; M. M. Bettman, D. D. S., Portland, Ore.; E. Melville Quinby, M. R. C. S., L. R. C. P., D. M. D., Boston, Mass.; Harold J. Leonard, D. D. S., B. A., and his secretary, Harriet A. Rice, A. B., M. D., of Columbia University, New York.

While every effort has been made to collect all the references bearing on the subjects which are included, it is recognized that there are probably omissions and errors. The Committee will appreciate it, if any that are noticed are brought to their attention, so that they can be noted in subsequent numbers. Such information should be sent to the Publication Office addressed to Grace Rogers Spalding, D. D. S., 555 West Maple Avenue, Birmingham, Michigan.

The Committee is indebted to Miss Inez Bowler, A. B., B. S., of the University of Michigan Dental College, for her aid in compiling the book list, and for helpful reference work and information.

Bibliography of Periodontia and References

Previous to 1700 A. D.

500 B. C.

Herodotus.

460 B. C.

Hippocrates. lib. 3 (p. 1082) De internis affectionibus.

50 A. D.

Pliny—C. Plinii Secundi. Historiae Mundi. 27 volumes.

131 A. D.

Galen, Claudius—Pergamus in Mysia, later Rome.

500 A. D.

"Leechdoms"—Saxon. "Caries and pyorrhea were not unknown."

Early 6th Century

Aetius—Greek physician of Arab birth.

9th Century

Rhazes—Persian. (Rasis opera, Venetiis, 1508).

10th Century

Avicenna—Arabian.

1300

Chauliac, Guy de—Avergne.

1597

Guillemeau, Jacques—Tr. by a Dutchman living at Oxford. The French Chirurgerie. Treatize of one hundred and thirteene diseases of the eyes and eye-liddes. 238 pp. London, 1622 (on p. 200. A discourse of the scorby), translated. Wyer's observations, also the same in Dutch: Hondert en dertien gebrekenen genesinge der oogen, en nu vermeerdert door Mr. Johannes Verbrigge. Amsterdam, 1678—232 pp.

1602

Foreest, J. Ziegler. Lugd. B. De aegiritudinibus laborium, gingivarum, dentium.

1687

Allen, Charles. London. 26 pp.—Curious observations on the teeth . . . The first known work on dentistry in the English language.

1691

Ruysch, Friedrich. Amsterdam. 138 pp. —Title: Friderici Ruyschii observationum anatomico-chirurgicorum, centuria, Amstelodami. 1691; Portal, op. cit. vol. III (Affirmed the relation between tartar and alveolar-pyorrhea).

1700-1800 A. D.

1723

Garengeot, René-Jacques. France. Transl. into German. Publ. in Berlin 1729.

1728

Fauchard, Pierre. Paris. 2 vols.—Le chirurgien dentiste, ou traité des dents.

1746

Bunon, Robert. Paris. 410 pp.—Experiences et démonstrations.

1753

Le Monnier, G. P. Paris. 90 pp.—*Dissertation sur les maladies des dents avec les moyens d'y remédier et de les guérir.

1757

Bourdet, Bernard. Paris. (1st ed.), (2nd ed. 1786), 2 vols.—Recherches et observations sur toutes les parties de l'art du dentiste.

1771

Auzébi, Pierre. Lyon. 167 pp.—Traité d'odontotalgie où l'on présente un système nouveau sur l'origine et la formation des dents, une description des différentes maladies, que affectent la bouche et les moyens de les guérir.

1772

Kunstmann. Stuttgart. (Describes removal of subgingival calculus)—Anweisung zur erhaltung gesunder und verbesserung kranker Zähne.

1775

Courtois, Honoré Gaillard. Paris. 343 pp. —Le dentiste observateur ou recueil abrégé d'observations tant sur les maladies qui attaquent les gencives et les dents, que sur les moyens de les guérir.

1778

Fandignère, Leroy de la. Nantes. 40 pp. —Manière de prévenir et guérir les maladies des gencives et des dents.

Hunter, John. London. 128 pp.—(Bd. with Natural history of human teeth.) A practical treatise on the diseases of the teeth.

Plenk, Joseph Jakob von. Vienna. 113 pp. —Doctrina de morbis dentium ac gingivarum.

1779

Bennett, William. London. 58 pp.—A dissertation on the teeth and gums and the several disorders to which they are liable.

1783

Woofendale, R. London. p. 97-132.—Practical observations on the human teeth.

1784

Ruspini, Bartholemeus. London. 96 pp.—A treatise on the teeth to which is added the most effectual method of treating the disorders of the teeth and gums.

1788

Jourdain, Berchillet Anselme Louis Bernard. (l. c. gives Bréchillet). 410 pp.—Traité des maladies et des opérations réellement chirurgicales de la bouche et des parties qui y correspondent.

1792

Carnelli. Naples. 16 pp.—Instruzione sopra le malattie delle gingive, e delli denti, e sopra la maniera di conservarli, con l'uso dell' elixir balsamico diseccativa, ed antiscorbutico.

1798

Mahon, Paul Augustin Oliver. Paris. 232 pp.—Le dentiste observateur ou le moyen de reconnaissance par la seule inspection des dents, la nature constitutive du temperament ainsi que quelques affections de l'âme; de garantir de souffrances cruelles et meme de la mort un grand nobre des enfans. Le tout suivi d'observations sur des maladies de sinus maxillaires, etc., et de differens avis interessans.

**Titles marked with an asterisk indicate dissertations or theses from universities in other countries.*

1800-1839 A. D.

1802

Longbothom, B. T. Baltimore. 66 pp.—Treatise on dentistry explaining the diseases of the teeth and gums with the most effectual means of prevention and remedy; to which is added dentition, etc.

1803

Fox, Joseph. London. 100 pp.—The natural history of the human teeth including a particular elucidation of the changes which take place during the second dentition and describing the proper mode of treatment to prevent irregularities of the teeth.

Serre, Johannes Jacob Joseph. Berlin. 564 pp.—Zahnarzneikunst. Praktische darstellung aller operationen der zahnarzneikunst. (Cited by Prinz.)

1804

Hirsch, Friedrich. Erfurt. 101 pp.—Bemerkungen ueber die Krankheiten des Zahnfleisches mit ohne Entzündung für Zahnärzte.

1805

Garlot, J. B. Paris. 162 pp.—Treatise on the diseases of the mouth, comprising the structure and functions of the mouth, the history of its diseases, the means of preserving it in beauty and health; and the operations pertaining to the dental art. English Ed. published in Baltimore 1842.

1806

Fox, Joseph. London. 170 pp.—The history and treatment of the diseases of the teeth, the gums and the alveolar processes with the operations they respectively require.

1810

Fuller, John. London. A popular essay on the structure, formation, and management of the teeth. p. 73-80.

1816

Aubry, Jean Baptiste Louis. Paris. 35 pp.—*Essai sur les maladies des gencives.

1817

Gerbaux, J. C. Edinburgh. 131 pp.—Gerbaux on the teeth . . .

1819

Bew, Charles. London. 126 pp.-95 pp., 1924.—Opinions on the causes and effects of diseases in the teeth and gums, with practical observations on the devastating consequences of their origin, as ascribable to lateral pressure. . .

1821

Koecker, Leonard. Philadelphia. 15 pp.—An essay on the devastation of the gums and the alveolar process. Reprinted from Phila. J. M. & Phys. Sc. No. 4, 1821, IV.

1822

Lemaire, Joseph Jean Francois. Paris. 3 vols.—Traité sur les dents; contenant la physiologie, pathologie et l'art opératoire.

Schumacher, Godofredus. Erfurt. 24 pp.—De morbis gingivarum.

1825

Sigmond, Joseph. Bath. 143 pp.—A practical and domestic treatise on the diseases and irregularities of the teeth and gums; with the methods of treatment.

1826

Koecker, Leonard. London. Principles of dental surgery.—p. 88-221, 270-99.

1828

Gutmann, S. Leipzig. Ueber die Behandlung der Zähne und de Zahnfleisches.

Jailloux, Louis. Paris. 29 pp.—*Sur les maladies des dents et des gencives.

1829

Fitch, Samuel Sheldon. New York. A system of dental surgery.—p. 213-48.

1830

Clark, John Paterson. London. A new system of treating the human teeth . . . ; 2nd ed. p. 44-68.

1831

Devaux-Bidon, Marie Pierre. Paris. 21 pp.—*Dissertation sur le stomatite en général spécialement sur la stomatite pseudo-membraneuse.

Winckworth, John. London. A popular treatise on the teeth and gums.

1832

Snell, James. Philadelphia. Practical guide to operations on the teeth.—p. 180-207.

1834

Imrie, William. London. Parents' dental guide.—p. 83-90.

Jobson, David Wemyss. Edinburgh. Outlines of the anatomy and physiology of the

teeth, their diseases and treatment.—p. 134-80.

Rudolph, Johannes Franz Valtin. Würzburg. 71 pp.—*Physiologisch und pathologisch semeiotische Betrachtung der menschlichen Zähne und des Zahnfleisches.

1835

American Journal of Dental Science. 160 pp.—The gums with late discoveries on their structure, growth, connections, diseases and sympathies. (Crowley gives the author of this as George Waite, Philadelphia, 1838, 80 pp. London 1835, 160 pp.)

Mallan, John. London. 146 pp.—Practical observations on the pathology and diseases of the teeth.

1836

Blume, E. Berlin. 207 pp.—Der praktische zahnarzt oder kurzgefasste lehre der zahnarzneiwissenschaft.

1838

Joullot, Jean Baptiste. Paris. 24 pp.—*De la suppuration simultanée des gencives et des alvéoles.

Lilling, Josephus. Praga. 19 pp.—De semeiotica dentium et gingivae.

Nordhausen, Fruhauf. Means of preserving and beautifying the teeth as well as the rapid and complete cure of all diseases of the teeth and gums by homoeopathic remedies.

Spooner, Shearjashub. Philadelphia. Guide to sound teeth. p. 136-56. 2nd ed.

Waite. See American Journal Dental Science. The gums; their structure; growth and connections.

Wardroper, William. London. 59 pp.—Structure, diseases and treatment of the teeth, considered with a view to the abolition in all common cases of the pernicious practice of tooth drawing.

1839-1900 A. D.

1839

Harris, Chapin Aaron. Baltimore. The dental art. p. 33-62, 221-63.

Hunter, John. Philadelphia. 127 pp.—Treatise on the natural history of the teeth explaining their structure, use, formation, growth and disease. (First publ. in 1771.)

Robertson, William. London. Practical treatise on the human teeth. p. 151-64.

1842

Hayden, Horace H. New York. On the diseases of the gums.

Visinet, E. Rouen. 257 pp.—Des gencives et des dents, de leurs maladies, des différents moyens thérapeutiques et hygiéniques propres à les en préserver on à les en guérir.

1843

Berdmore, Thomas. Baltimore. 87 pp.—Treatise on the disorders and deformities of the teeth and gums, explaining the most rational methods of treating their diseases.

Maury, J. C. F. Baltimore. Treatise on the dental art. p. 91-155.

1846

Grapin, Jean Baptiste Antoine. Paris. 60 pp.—De la stomatite mercurielle.

1850

Buuren, Van Bernardus Gronigen. 44 pp.—*De stomatocace.

1851

Bergeron, Rémi Louis. Paris. 31 pp.—*De la stomatite en général et en particulier de la stomatite ulcéreuse chez les soldats.

1853

Boyaux, J. N. Paris. 22 pp.—Du gonflement chronique et fongueux des gencives.

Howard, Thomas. London. 93 pp.—On the loss of the teeth and on the best means of restoring them, with a new and improved method of fastening loose teeth.

1855

Jardel, J. F. Dijon. De l'absorption des gencives et des alvéoles.

Messier, Leon. Paris. 34 pp.—*De la stomatite ulcéro-membraneuse.

1857

Bruck, J. Leipzig. 77 pp.—Die scrofulöse zahnaffection.

Hutchinson, David. Philadelphia. 18 pp.—What are the causes (and nature) of that disease, incident to pregnancy and lactation, characterized by inflammation and ulceration of the mouth and fauces, usually accompanied by anorexia, emaciation and diarrhoea; and what is the best mode of treatment? (Repr. from A. J. M. Sci. 1857 n. s. xxx.)

1859

Bergeron, Étienne Jules. Paris. 252 pp.—De la stomatite ulcéreuse des soldats et de son identité avec la stomatite des enfants dites couenneuse, dipthéritique, ulcéro-membraneuse.

1860

Richardson, Benjamin Ward. London. 274 pp.—On the medical history and treatment of diseases of the teeth and the adjacent structures. (Lectures delivered 1858-9.)

1861

Blondeaux, E. J. Paris. 44 pp.—*De la stomatite ulcéreuse des enfants et de la nécrose du maxillaire dans cette affection.

Delestre, Gustave. Paris. 56 pp.—*Du ramollissement des gencives.

1862

Albrecht, Eduard. Berlin. 90 pp.—Klinik der mundkrankheiten.

1862

Hulme, Robert Thomas. London. 76 pp.—Contributions to dental pathology. Calcification of the dental pulp. Three lectures on diseases of the dental periosteum.

1863

Péchaud, Jacques, Joseph. Strasbourg. 34 pp.—*De la stomatite ulcéreuse spécifique chez les soldats en particulier.

1864

Cacciaguerra, Matteo. Catania. 29 pp.—Modificazione sulla legatura dei denti.

Pansch, Adolf. Halis, Sax. 29 pp.—*De pyorrhoea alveolari seu gingivitide expulsiva.

1865

Bertin, Auguste. Paris. 311 pp.—Traité du déchaus sement et de l'enbranlement des dents et des maladies des gencives.

Putegnat, Joseph Dominique Ernest. Paris. 32 p.—De la stomatite gangréneuse.

1866

Pretérre, Pierre Apollonie. Paris. Les dents. p. 81-96, 104-7.

Thevenin. Paris. *Stomatite ulcéreuse des enfants.

1867

Bruck, Julius, Jr. Leipzig. 60 pp.—Die Krankheiten des Zahnfleisches.

Magitot, Emile. Paris. 34 pp.—Mémoire sur l'ostéopériostite alvéolodentaire. (Repr. from Arch. gén. de méd, 1867. 6. s., IX.)

1868

Heider, Moriz and Wedl, Carl. Leipzig. Atlas zur Pathologie der Zähne.

1869

Vergne, Alfred. Paris. 55 pp.—*Sur tartre dentaire et ses concrétions.

1870

Wedl, Carl. Leipzig. 362 pp.—Pathologie der Zähne.

1871

Arthur, Robert. Phildelphia 256 pp.—Treatment and prevention of decay of the teeth.

Harris, Chapin Aaron. 10th ed. 794 pp. 1885—11th ed. 994 pp.—Principles and practice of dentistry.

1872

Corbetta, Giovanni. Milano. 334 pp.—Nuovo trattato igienico e curativo dei denti e delle gengive. 2nd ed.

Martin, Dupont, Ferdinand. Paris. 40 pp.—De la gingivite ulcéreuse des matelots.

1876

Pietkiewicż, Valérien. Paris. 207 pp.—De la périostite alvéolodentaire.

1877

Baronnet, Jules Albert. Paris. 54 pp.—*Cause anatomo physiologieque d'ostéo-periostite alvéolo-dentaire jeunes sujets et chute prématurée des dents consécutive.

Pinard, Adolphe & Pinard, Désiré. Paris. 26 pp.—De la gingivite des femmes enceintes et de son traitement.

Rehwinkel, F. H. Chicago. Pyorrhea alveolaris. A. D. A. Trans. p., 96.

1878

Arguello, Joaquin. Paris. 36 pp.—*Des stomatites fétides dans las intoxication par le plomb, l'arsenic et le phosphore.

Chaillou, J. Paris. 46 pp.—*Étude du liséré gingival dans certaines impregnations métalliques.

Mills, George A. New York. Series of articles of the so-called Riggs' disease (loosened teeth or pyorrhea alveolaris (pus discharges from the sockets of the teeth.)

Rousseau, Henri. Paris. 116 p.—De l'acide

chromique . . . (Use for gingival infections.)

1879

Maget, Lucien Gabriel. Paris. 54 pp.—*Étude sur l'étiologie de la stomatite ulcéreuse des soldats et des marins.

1880

Bontems, Victor. Paris. 76 pp.—*De la gingivite. Essai de classification. Ses formes. Son traitement.

Guébey, J. Paris. 42 pp.—*Étude sur l'ostéo-periostite alvéolaire (ostéo-périostite alvéolo-dentaire de Magitot.

Scheaff, Julius, Jr. Vienna. 416 pp.—Lehrbuch der Zahnheilkunde für praktische Aerzte und studirende.

1881

Coutemoine, Élisée. Paris. 33 pp.—*Contribution à l'étude de la stomatite ulcéromembraneuse chez les personnes âgées (depuis 40 ans.)

Magitot, Emile. Paris. p. 248-98.—Recherches sur les gencives et leurs maladies (Extrait du Dictionnaire encyclopédique des sciences médicales. Article: Gencives.)

1882

Mills, George A. New York. 8 pp.—Pericementitis: its manifestations in the oral cavity and its serious effects upon the general health.

1883

Alberti. Paris. (see Weinberger) *Pathogénie et traitement de la gingivite expulsive; suppuration des alveoles; chute prématurée des dents; ostéo-périostite alvéolodentaire.

Didsbury, Henry. Paris. 42 pp.—De l'état des gencives chez les femmes enceintes et de son traitement.

1885

Arkövy, Joseph. Stuttgart. 404 pp.—Diagnostik der zahnkrankheiten und der durch zahnleiden bedingten kiefererkrankungen.

Leip. Vogel. 506. p.

1886

Helmkampff, Hermann. Stuttgart. 248 pp.—Diagnose und Therapie der Erkrankungen des Mundes und Rachens sowie der Krankheiten der Zähne.

1887

Black, Greene Vardiman. Chicago. 138 pp.—A study of the histological characters of the periosteum and peridental membrane.

Litch, W. F. Philadelphia. 2 vols. Entered as "American System of Dentistry" . . . ed. by Wilbur F. Litch.

1888

Blodgett, Albert Novatus. Philadlephia. 279 pp.—A handbook of dental pathology.

Galippe, M. L. V. Berlin. 30 pp.—(Hft. 1 of Abhandlungen a. d. zahnärztl. praxis.) *Die infectiöse arthro-dentäire gingivitis (pyorrhoea alveolaris).

Vi(n)centini, Filandro. Italy. 1888 and 1893. Sulla diagnosi botanico della "leptotrix racemosa" . . .

1889

Brucke, Carl Adolf Max. Berlin. 31 pp.—Ueber idiopathische gangränöse stomatitis.

Fillebrow, Thomas. Philadelphia. 273 pp.—Text-book of operative dentistry.

1890

Catching's Compendium of Practical Dentistry. Atlanta, Ga.

Miller, Willoughby Dayton. Philadelphia. 364 pp.—Micro-organisms of the human mouth.

Richer, P. Paris. *De la périodontite expulsive et de son traitement.

Schech, Phillip. Leipzig. 351 pp.—Die krankheiten der mundhöhle des rachens und der nase. 3 aufl.

1891

Barillé, Alexandre. Paris. 74 pp.—*Des complications infectieuses de la périodontite suppurée.

Richter, H. E. Milwaukee. Two cases of ulcero-membranous gingivitis described. How we may preserve our youth.

1891

Schmidt, Hermann Karl Wilhelm. Greifswald. 25 pp.—Über gingivitis gangraenosa.

1892

Abbott, Frank. Philadelphia. 240 pp.—Dental Pathology and practice.

1893

Maurel, Edward César-Emile. Paris. 399 pp.—Traité des maladies de la bouche

(pathologie interne), suivi d'un précis d'hygiene de cette cavité.

Rauchfus. (?). Demonstration of B. fusiformis and Borrelia in ulcero-membranous gingivitis (cited by Tarassiewicz).

1894

Cravens, Junius E. Indianapolis. 48 pp. —A treatise on pyorrhea alveolaris (Riggs' disease); a system for its prompt, positive and permanent cure.

1895

Chompret, Joseph. Paris. 90 pp.—Essai sur les gingivites infectieuses.

Hopewell-Smith, Arthur. Philadelphia. 119 pp.—Dental microscopy.

Nash, Henry S. N. Y. 2 vols.—Loosening teeth, or chronic alveolitis (pyorrhoea alveolaris, phagedenic pericementitis, Riggs' disease, etc.); Its causes, clinical history and treatment; with general directions for care of the teeth.

1896

Frey, Leon (and Ruppe, Ch.). Paris. 279 pp.—Pathologie des dents et de la bouche.

Herbst, Wilhelm. Berlin. 296 pp.—Methoden und Neuerungen auf dem Gelisete der Zahnheilkunde.)

1897

Bernheim. (De Sotolongo, Habana, Cuba). 126 pp.—Treatise, 1910. 95 pp.—Report on thirty cases of Vincent's.

Chieti. Also Bacteria of the sputa and cryptogamic flora of the mouth. London, 232 pp.

Montell, William A. 8 pp.—Pyorrhea alveolaris.

Younger, William J.

1898

Lehedinsky, J. Paris. 66 pp.—Les gingivo-stomatitis et le polymicrobisme buccal.

Smale, Morton and Colyer, J. F. 423 pp. —Diseases and injuries of the teeth. p. 286-311.

Zentler, Arthur. *Extraction in acute suppurative pericementitis.

1899

Eames, George Franklin. Philadelphia. 240 pp.—Practice of dental medicine.

Fitzgerald, John. London. 62 pp.—Pyorrhea alveolaris and its relation to general medicine.

Römer, Oskar. Freiburg. 50 pp.—Zahnhistologische Studie.

Talbot, Eugene Solomon. Philadelphia. 192 pp.—Interstitial gingivitis, or so-called pyorrhoea alveolaris.

1900-1931 A. D.

1900

Lesueur, Louis. Paris. 97 pp.—Recherches sur la stomatite ulcéro-membraneuse; l'angine ulcéro-membraneuse à bacilles fusiformes et spirilles et leur analogie.

Morhange, Gustave. Paris. 64 pp.—*Contribution à l'étude de la gingivite (expulsive) et de son traitement.

1901

Barrett, William C. Philadelphia. 319 p. —Oral pathology and practice. 2nd ed.

1903

Courchet, Pierre. Paris. 72 pp.—Traitement de la pyorrhée alvéolo-dentaire.

Grünwald, Ludwig. Philadelphia. 219 pp. —Tr. by Newcomb. Atlas and epitome of diseases of the mouth, pharynx and nose. 2nd ed.

Montigny, Ornbrose de. Paris. 64 pp.—*Etude clinique sur l'angine de Vincent.

1904

Smith, D. D. Philadelphia. 18 p.—Alveolar pyorrhea; its cause, sequelae and cure.

1905

American textbook of operative dentistry . . . ed. by Edward C. Kirk; 3rd ed. 899. pp.

Léger-Dorez. Tours. Le gaz oxygène dans le traitement de la pyorrhée alvéolo-dentaire et les trajets fistuleuses.

Nesmiezanowa, L. Moscow. Pyorrhea alveolaris.

1906

Abbadie, Victor. Paris. 52 pp.—Traitement de la pyorrhée alvéolo-dentaire.

Goadby, Sir Kenneth Weldon. London. 44 pp.—Vaccine treatment of pyorrhoea alveolaris.

Preiswerk, Gustav. Philadelphia. 343 p.

—Atlas and text-book of dentistry including diseases of the mouth.

Roque, Germain & Galliard, L. Paris. 276 pp.—Maladies de la bouche, du pharynx et de l'oesophage.

Tomes and Nowell. Dental Surgery. pp. 636.

1908

Broomwell, Isaac Norman. Philadelphia. 496 pp.—Practical dentistry.

1909

Guerini, Vincenzo. Philadelphia. 355 pp. —History of dentistry.

Lagarde, Maurice. Paris. 98 pp.—*La stomatite mercurielle maladie fuso-spirillaire (étude bactériologique et critique).

Marshall, John Sayre. Philadelphia. 717 pp.—Manual of the injuries and surgical diseases of the mouth and jaws. 3rd ed.

1910

Colyer, Sir James Frank. London. 1000 pp.—Dental surgery and pathology. (4th ed. N. Y. 1919. 889 pp.)

Fourquet, Elie. Paris. 324 pp.—Les arthrites alvéolo-dentaires.

Johnson, Charles Nelson. Philadelphia. 769 pp.—Text-book of operative dentistry. 2nd ed. Article on Pyorrhea pp. 455-475.

Malassez, Luis Charles. Paris. 268 pp.—(and Galippe, Marie, Louis-Victor). Les débris épithéliaux paradentaires.

1911

Blessing, Georg. Leipzig. 42 pp.—Zur Bakteriologie und antibakteriellen Therapie der Pyorrhea alveolaris.

Colyer, James Frank and Colyer, Stanley. London. 189 p.—Dental disease in its relation to general medicine.

Nogue, Raymond and other. Paris. 430 pp.—Maladies paradentaires. (v. 5 of Traité de stomatologie).

Tellier, Julien. Lyon. La pyorrhée alvéolaire.

1912

Engs, John S. 8 pp.—Pyorrhea alveolaris.

Pickerill, Henry Percy. London. 268 pp. —Stomatology in general practice.

Neuman, Robert. Berlin. 69 pp.—Die alveolar-pyorrhoe und ihre Behandlung. Erfahrungen und therapeutische Winke aus der Praxis.

Noguchi, Hideyo. *Treponema mucosum (new species) a mucin-producing spirocheta from pyorrhea alveolaris grown in pure culture.

Witkowski. London. 54 pp.—The tightening of loose teeth. Some technical innovations. Tr. by H. & G.

1913

Hecker, Friedrich. St. Louis. 157 pp.—Pyorrhea alveolaris.

Stenhouse, John; Ferguson, John. Philadelphia. 278 pp.—Pathology, general and special. 2nd ed.

Talbot, Eugene Solomon. Toledo. 340 pp. —Interstitial gingivitis and pyorrhoea alveolaris.

1914

Bennett, Norman G. Science and practice of dental surgery. p. 487-513.

Euler, Hermann & Graser. 2 vols.—Behandlung der Erkrankungen der Zähne und des Zahnfleisches. (In Penzoldt-Stintzing, Handbuch der ges, therapie. 5 auf.)

Francois, Jules. Paris. 127 pp.—L'ostéomyélite aiguë du maxillaire supérieur chez le nourrisson.

Pickerill, Henry Percy. London. 374 pp. —Prevention of dental caries and oral sepsis. (Calculus, p. 303; pyo. alv. p. 27.)

Renaux, A. F. A. Paris. *La symbiose fusospirillaire et son traitement par l'arsénobenzol.

1915

Adair, Robin. Atlanta, Ga. 462 pp.—Practical oral hygiene, prophylaxis and pyorrhea alveolaris. 2nd ed.

Bass, Charles C. and Johns, Foster M. Philadelphia. 167 pp.—Alveolodental pyorrhea.

Black, Greene Vardiman. Chicago. 489 pp.—A work on special dental pathology.

Llewellyn and Jones. N. Y. 693 pp.—Fibrositis.

Smith, A. J. and M. T. Barrett. 160 pp. —A review of recent studies upon amoebic pyorrhea. (Periodical articles.)

1916

Colyer, Sir James Frank. London. 102 pp. —Chronic general periodontitis.

1917

Goodey, T. and Wellings, A. W. Obser-

vations on endameba gingivalis from the human mouth. (Parasitology v. 9 p. 537-59.)

Hall, J. T. London. Suggestions for pyorrhea patients during and after ionization treatment.

Sachs, H. Paradentitis und paradentose (die sog. pyorrhoea alveolaris). In Handbuch der zahnheilkunde. Bd. 2 s. 454.

1918

Grief, Samuel. New York. 71 pp.—(History of dentistry); dentistry in the Bible and (the) Talmud.

Hopewell-Smith, Arthur. Philadelphia. 2 vols.—Normal and pathological histology of the mouth.

Keyes, Frederick Anthony. New York. 504 pp.—Army dentistry. Forsyth Lectures.

1919

Beyer, E. Berlin. 63 pp.—Die Alveolarpyorrhöe als lokale Spirochätose der Mundhöhle. Aetiolgie und Therapie.

Cahier, Ferdinand Eugene Gaetan. Paris. 62 pp.—*Le traitement des localizations buccopharyngées de l'infection fuso-spirillaire (stomatite ulcéreuse, stomatite mercurielle, angine de Vincent) (par l'arsénobenzol 606 et 914).

Dobell, Clifford. New York. 155 pp.—Amoebae living in man.

Reinhold, Walter. Berlin. 21 pp.—Ueber die moderne Behandlung der alveolarpyorrhöe.

Sturridge, Ernest. Philadelphia. 139 pp.—Periodontal disease and its treatment by ionic medication.

Seidel, W. H. Die beziehungen der sperochäten and der salvarsantherapie zu pyorrhoea alveolaris und anderen erkrankungen des Mundes. (Deutsche zahnheilkeude No. 41.)

Ziesel, William. Charlotte, N. C. 19 p.—Pyorrhea extermination (gingivo-ectomy).

1920

Braun, C. Berlin. Fibromatöse Entartung des Zahnfleisches.

Crow, Douglas, Arthur. London. 111 pp.—Pyorrhea alveolaris in its clinical aspect.

Filleul, Charles. Paris. 54 pp.—*Contribution à l'étude des stomatites.

Garth, R. Leipzig. *Ein Beitrag zur Therapie der Alveolar-pyorrhöe mit besonderer Berücksichtigung der Befestigungschienen.

Gottlieb, B. (und Fleischmann) Berlin. 16 pp.—Beiträge zur Histologie und Pathogenese der Alveolar-pyorrhöe.

Head, Joseph. Philadelphia. 382 pp.—Modern dentistry. 2nd ed.

1921

Bachmann, R. Leipzig. *Ueber die *Stomatitis ulcero-membranosa.*

Baerwolf, F. E. Berlin. *Therapeutische Erfolge bei Vaksinetherapie und Protoplasmaaktivierung der pyorrhoischen Diathese.

Black, Arthur D. Buffalo, N. Y. Index to Dental Periodical Literature.

Dental Federation of Japan. The present condition of dentistry in Japan.

Hille, A. Leipzig. Zur Kenntniss der Alveolarpyorrhöe.

Kappauf, Willy. Mannheim. 80 pp.—Aus der Zahnheilkunde der Araber . . . . (Dentistry of the Arabs).

Lewin, Hugo. Berlin. 26 pp.—Zahnerkrankungen und deren Behandlung im alten Aegypten.

Merritt, Arthur H.. New York. A brief history of periodontology. (In Journal of dental research. v. 3, p. 146.)

Oy, Hugo von. Leipzig. 20 pp. The pathohistologic alterations in the gingiva (In German—Die pathologisch histologischen Veränderungen am Zahnfleisch . . .)

Pont, Albéric. Paris. 898 pp.—Précis des maladies des dents et de la bouche. 2nd ed. 1929, 798 pp.

Ruffer, Sir Marc Armand. Chicago. 327 pp.—Studies in the palaeopathology of Egypt.

1922

Bouvet, Pierre. Paris. 105 pp.—Les lésions dentaires des hommes préhistoriques.

Ducournav, Albert. Paris. 294 pp.—Dents et maux des dents.

Hahn, Magdalene. Germany. 34 pp.—*Konstitution und Alveolarpyorrhöe.

Kranz, Peter and Falck, Karl. Berlin. 172 pp.—Alveolar-pyorrhöe; ihre Aetiologie, Pathologie und Therapie. . . .

Marcovici, Alexandre. Paris. 36 pp.—*De l'emploi du paraamino-benzoate de butyle normal en stomatologie.

Römer, Oskar. Periodontis chronica marginalis purulenta. Pyorrhea alveolaris. (In Scheff, Julius ed. Handbuch der zahnheilkunde. 3 aufl. Bd. 2, 1 abt. Wien, Hölder, 1922.)

Stillman, Paul R. and McCall, John Oppie. New York. 240 pp.—Textbook of clinical periodontia.

Straubel, Paul Hermann. Leipzig. 24 pp. —Zahn und Mundleiden und deren Behandlung bei Paulus von Aégina. (About 610-641 A. D.)

Zehrfeld, A. Leipzig. *Terpichinininjektionen bei Stomatitiden.

1923

Berger, Adolph. Brooklyn. 450 pp.—Principles and technique of oral surgery.

Dental Board of the United Kingdom. (Great Britain) London. 96 pp.—Lectures on "The diseases of the periodontal tissues due to infection in their relation to toxemia."

Goadby, Sir Kenneth Weldon. London. 383 pp.—Diseases of the gums and oral mucous membrane.

Hirschfeld, Isador. *A study of skulls in the Amer. Mus. of Nat. Hist. in relation to periodontal disease—published in Journal of dental research, vol. 5, p. 241-65.

Price, Weston A. Cleveland. 2 vols.—Dental infections. See vol. 1.

Wilker, Hermann Heinrich. Dresden. 40 pp.—Der Arzt Johan Stephen Strobelberger, mit besonderer Berücksichtigung seines Werkes: De dentium podagra. 1630.

1924

Box, Harold Keith. Toronto, 120 pp.—*Studies in periodontal pathology. (Canadian dental research fnd. Bull. 7)

Christ, Walter. Frankfurt. Veränderungen an der Alveole bei unnatürlicher Belastung, ein Beitrag zur Belastungs mechanik der Zähne.

Gallian, Roger. Paris. 140 pp.—Hygiène et prophylaxie bucco-dentaires.

Malleson, Herbert Cicil and Bulleid, Arthur. London. 72 pp.—Practical dental histology and bacteriology. A manual for students.

Moral, Hans and Frieboes, Walter. Leipzig. Atlas der Mundkrankheiten.

Jomini, E. C. F. Zürich. Contribution a l'étude expérimentale du vaccin de Goldenberg dans la pyorrhée alvéolarie.

Neumann, Robert. Berlin. 170 pp.—Die Behandlung der Sogenannten Alveolar pyorrhoe und der anderen Paradentosen.

Nouel, Maurice. Caracas. 45 pp.—*"Anotaciones sobre la piorrea alveolo-dentaria."

1925

Appleton, Joseph Luke T., Jr. Philadelphia. 474 pp.—Bacterial infection with special reference to dental practice.

Bazin, L. Paris. *Des syndromes généraux consécutifs à la pyophagie d'origine dentaire du traitement vaccinal de la pyorrhée et de ses complications. (Vaccin du Dr. Goldenberg.)

Bourgeois, (Mme.) M. M. *Contribution à l'étude du traitement de la pyorrhée alvéolaire.

Brown, O. M. Denver, Colo. The truth about pyorrhea and its treatment.

Coriat, Maurice. Paris. 48 pp.—De la doctrine dentaire de la stomatite ulcéromembraneuse et de l'angine de Vincent.

Gande, Bruno. Berlin. 2nd ed.—Die Spirochüten der menschlichen Mundhöhle nebst einer Einführung in die mikroskopische Technik.

Gerber, F. Bienne. *Des gingivo-stomatites.

Gottlieb, Bernard. Berlin. 88 pp.—Schmutzpyorrhoe, Paradental-pyorrhoe und Alveolaratrophie; Klinik, Aetiologie, Prophylaxe und Therapie.

Heimbrod, F. Geneva. La pyorrhée advëolodentaire; son traitement par le stovarsol.

Kells, Charles Edmund. St. Louis, Mo. 510 pp.—The Dentist's own book.

Moorhead, Frederick Brown and Dewey, Kaethe Weller. Philadelphia. 540 pp.—Pathology of the mouth.

Reinmöller, M. Max. Berlin. 45 pp.—Klinische Untersuchungen ueber Alveolarpyorrhöe.

Römer, Oskar (In D. Zahnhlk Hft. 66). Die pathologisch-anatomischen Grundlagen der sog. alveolar-pyorrhoe.

Welge, Walter. Hannover. 27 pp.—*Zur Behandlung der Gingivitis ulcerosa.

1926

Broderick, Ralph A. Edinburgh. 143 pp.

—Dental bacteriology. (Outlines of dental science, v. 2.)

Burchard, Henry H. and Inglis, Otto Edward. Philadelphia. 879 pp.—Textbook of dental pathology and therapeutics for students and practitioners.

Cohnen, Grete. Berlin. *Zur Therapie der *stomatitis ulcerose.*

Colyer, Stanley. New York. 75 pp.—Chronic infections of the jaws.

Guttmann, Georg. Berlin. 163 pp.—Haut- und Infektionskrankheiten mit besonderer Berücksichtigung ihrer Erscheinungen in Munde. Ein Leitfaden für Zahnärtze und Studierende.

Heddema, Engelhard, Hermann. Berlin. 40 pp.—*Alveolarpyorrhöe.

Kells, Charles Edmund. 563 pp.—Three score years and nine.

Lake, Frederick W. 112 pp.—Ultra-violet radiation in dental pathology.

Marcovici, Gabriel. Paris. 51 pp.—*Contribution à l'étude des gingivo-stomatites.

Marshall, John Albert. Philadelphia. 331 pp.—Diseases of the teeth.

Neumann, Robert. Berlin. Atlas zur radical-chirurgischen Behandlung der Paradentosen.

Regis, Bazin. *Contribution à l'étude des pyorrhées alvéolaires—essai critique sur la vaccinothérapie locale comme mode de traitement de ces affections.

Sarthou. Bordeaux. *De l'étiologie et du traitement de les pyorrhée alvéolaire.

Siegmund, Herbert and Weber, Rudolf. Leipzig. 330 pp.—Pathologische Histologie der Mundhöhle.

Ward, Marcus Llewellyn (ed.) Philadelphia. 818 pp.—Amer. textbook of operative dentistry in contributions by eminent authorities (6th ed.)

1927

Buxton, J. L. Dudley. New York. 193 pp.—Dental pathology (Outlines of dental science, vol. 7.)

Endelman, Julio. London, 444 pp.—Special dental pathology. 2nd. ed.

Euler, Herman. Munich. 353 pp.—Pathohistologie der Zähne mit besonderer Berücksichtigung der Pathobiologie.

Fones, Alfred. Philadelphia. 348 pp.—Mouth Hygiene, 3rd ed.

Haupl, Karl and Lang, Frank Joseph. Berlin. 404 pp.—Paradentitis marginalis. Ihre Pathologie, Aetiologie, Klinik, Therapie u. Prophylaxe etc., etc.

Heinrich, Oskar. Berlin. 94 pp.—Die Alveolarpyorrhoe und ihre Behandlung sowie die wieder Befestigung lockerer Zähne unter besonderer Berucksichtigung für den Dentisten. (In Fachbücherei der dentisten Bd. 7.)

Hommens, Eugen. München. 29 pp.—Untersuchungen über die histologischen Veränderungen der Wurzelhaut bei *periodontitis acuta.*

Imbert, A. Paris. Le problem du déchaussement des dents.

Imrie, David A. Lausanne. 23 pp.—L'infection des dents et des os maxillaires (son interprétation radiographique et microscopique) avec un ces d'ostéite déformante.

Kiefer, E. Berlin. 54 pp.—Befestigungs- und Entlastungsschienen.

Korden, Fritz. München. 29 pp.—Untersuchungen des Blutbildes bei Gingivitiden unter besonderer Berücksichtigung der gingivitis ulcerosa.

Neuman, Robert. Berlin. 17 pp.—Radikalchirurgischer Behandlung der Paradentosen (alveolar-pyorrhöe). (Tr. in Ger., Eng., Fr., It., Rus. and Sp.)

Möllendorff, Wilhelm von, ed. Berlin. 7 vols.

Steadman, Sidney Francis St. Jermain. London. 263 pp.—Pyorrhea alveolaris.

Vilar, Fiol R. Barcelona. 111 pp.—La piorrea alveolar. Compendio practico sobre etiología, terapéuti ca y tratamiento.

Wolff und Stock. 2 auf. 88 pp.—Die Wiederbefestigung lockerer Zähne.

1928

Box, Harold Keith. Toronto. 123 pp.—The treatment of the periodontal pocket (Canadian dental research fnd. Bull. 10).

Broderick, Frederick William. London. 364 pp.—Dental medicine.

Burkhart, Harvey Jacob. New York. 49 pp.—Care of the mouth and teeth.

Eyer, Federico. Rio de Janeiro. Piorréa alveolar e son tratamento moderno.

Goll, Hermine. Heidelberg. Paradentosen. Pathologisch-anatomische Veränderungen.

Haden, Russell Landram. Philadelphia. 165 pp.—Dental infection and systemic diseases.

Keller, Konrad. Zurich. 64 pp.—*Die akute und chronische Periodontitis und ihre Behandlung mit Septojod.

Leigh, Rufus W. Berkeley, p. 399-440.—Dental pathology of aboriginal California. (V. of Cal. pub. in Am. Archaeology and ethnology. v. 23, No. 10.)

Luthi, Rudolf. Berlin. pp. 96-110.—*Ueber den einfluss von chlorphenol, phenol, tricresol-formalin, triopaste, Albrechtsche Wurzelfüllung und Eugenol auf das Periodontium des Mundes.

Mead, Sterling Vernon. St. Louis. 2nd ed. 578 pp.—Diseases of the mouth.

Orban, Balint. Chicago. 223 pp.—Dental histology and embryology.

Ottolengui, Rodrigeus. Brooklyn. 488 pp.—Table talks on dentistry.

Prinz, Hermann. Philadelphia. 248 pp.—Diseases of the soft structures of the teeth and their treatment.

Schwartz, Ludwig. Berlin. p 513-30.—*Zur aetiologie und Pathogenese der Paradentose.

Sorrin, Sidney and Samuel Miller. New York. The practice of periodontia.

Tanguy, Thos. Victor. St. Louis. 111 pp.—The science and practice of dental actinotherapy.

Walkhoff, Otto. Berlin. 127 pp.—Mein System der medikamentösen Behandlung schwerer erkrankungen Zahnpulpa und des Periodontiums.

Weissenbruch, Adolf. Darmstadt. 20 pp.—*Die klinischen Diagnose apikaler und marginaler Paradentitiden.

1929

Bunting, W. Russell. Philadelphia. 495 pp.—Textbook of oral pathology for students and practitioners of dentistry.

Cahn, Lester Richard. New York. 152 pp.—The basic principles of general and oral pathology.

Delater, Gabriel. Paris. Les processus inflammatoires et tumoraux peridentaires; étude bacteriologique et histologique des suppurations alvéolodentaires, des pyorrhées, des granulomes et kystes paradentaires des epithéliomes adamantins des epulis.

Greve, Hans Christian. 10 auf. p. 56-87. Diagnostisch-therapeutisches Taschenbuch für Zahnärzte.

Münch, Eduard. Quakenbrück. 34 p.—*Seitliche Belastung als ätiologisches Moment bei der Entstehung der Alveolarpyorrhoe.

Omnès, Henri. Paris. 60 pp.—Contribution à l'étude de la pyorrhée alvéolaris. Le rôle des troubles ovariens dans la production de l'alvéolyse.

Ottolengui, Rodrigues. Outlines of preventive medicine. See p. 269.

Port, Gottlieb, and Euler. Munich. 4th ed. 767 p.—Lehrbuch der Zahnheilkunde.

Sachs, Hans. 220 pp. Die Behandlung lockerer Zähne nach Younger—Sachs.

Wallace, J. Sims. London. Physiology of oral hygiene and recent-research.

1930

Box, Harold Keith. Toronto. 63 pp.—Necrotic gingivitis, (trench mouth). (Canadian dental research foundation. Bull. 14.)

Kiefer, E. Die Behandlung der Paradentose durch Schienen und Entlastungsbügel. (In preparation Meussen.)

Kühn, Dr. Alfred. Leipzig. 57 pp.—Einführung in die pathologische Histologie der wichtigsten Pulpa-und Kiefererkrankungen sowie der hauptsächlichsten Tumoren der Mundhöhle. (Section on Alveolarpyorrhoe und die Osteo-myelitis.)

McAsh, Ian Fleming. London. 290 pp.—A dictionary of dental diseases and treatment.

Merritt, Arthur H. New York. 289 pp.—Periodontal diseases.

Ottofy, Louis. Fifty years in dentistry.

Pellkofer, Josef. Tübingen. 31 p. *Perkutane Behandlung der Gingivitis ulcerosa mit Dermaproten.

Stärke, W. Die pratheitik im dienste der paradentose behandlung.

Zemsky, James L. Brooklyn. 404 pp.—Oral diseases. See pp. 316-330, also 323 (sections).

(a)—Periodontal Histopathology

1927

1—Beckwith, T. D. with Williams and Fleming. **Regeneration of rodent peridental membrane.** Proc. Soc. Exper. Biol. and Med., Mar., 1927. Abs. D. Cosmos lxix 755. July, 1927.

A dental bur was passed through the gum and alveolar process into the dentin of the permanent growing incisor of rabbits and rodents. The animals thus treated were sacrificed at semi-weekly intervals over a period of six weeks and the histological changes of the inner part studied. A point of special interest lies in the fact that repair of the periodontal tissue is begun at the tooth surface rather than at that of the alveolar bone.

Although the abstractor is willing to admit the interest of this observation, he must point out that, in the rodent incisor, there is no connection between the intra-alveolar surface of the tooth and the periodontal tissues, and that therefore these findings can have only a remote bearing on the question of repair of periodontal injuries in the human being. —*Copy.*

2—Brash, J. C., M. C., M. A., M. D.: **The growth of the alveolar bone and its relation to the movements of the teeth, including eruptions,** in: Dental Record (December, 1926), January, 1927.

Observations recorded in this valuable contribution were made on pigs, fed madder, and, among others, the following conclusions reached: (1) That alveolar growth is the main if not the only factor in growth depth of the body of mandible and maxilla and continues throughout growth period of jaws or practically till 3rd molars have erupted. (2) Along with general growth come additions to the walls of alveoli causing teeth, both erupted and un-erupted to advance towards functional position. (3) The proximate cause of tooth eruption is an intensification locally of the general upward growth of alveolar bone. (4) Growth of cementum is also a factor, tho a lesser one.

The general conclusion is that the alveolar arches are in a constant state of change. Not only is there constant upward growth, the actual plane of the alveolar border carrying the teeth with it being constantly renewed, but there is also a continuous movement of all the teeth in two other directions —buccal and proximal. The actual position of the teeth in the arch at any given time is the resultant of these three movements. Differential rates of movement in these three directions will bring about the changes to be observed in the normal development of the alveolar arches and a disturbance of these movements, however caused, will readily account for anomalies in the shape of the arches and in the position of the teeth.

The alveolar arches can no longer be regarded as stationary and unchanging, unaffected by the growth of bone, save only in minor degree and locally, as teeth in different places are shed and replaced. They must be considered on the contrary, as in a constant state of change and reformation. The alveolar arch of the permanent dentition is quite a different thing from the arch of the deciduous dentition, the bone of which it is composed being entirely new, and although it may resemble the deciduous arch in general form and dimensions plus the added portions behind, yet it is not merely the deciduous arch changed a little in front and extended, but a new formation which has come into existence by the mechanism of bone growth here described, and in conformity with the general growth of the jaws of which it is an integral part.

(See D. Cosmos LXIX—1927, 1074-5 for more extended review.)

—*C. R.*

3—Cahn, Lester R., D. D. S.: **The pathology of pulps found in pyorrhetic teeth,** in: D. Items of Interest. xlix. August, 1927.

The periodontal membrane dips into the pulp canal as far up as the cemento-dentinal junction and overlaps at this junction or is intertwined with the pulp. This close proximity of the two tissues readily shows how the pulp can become both contaminated and infected. An infective process rising in the so-called "pyorrhetic pocket" can easily spread along the periodontium to the apex,

resulting in pulp involvement. A series of experimental studies were made by scaling the apical foramina, as well as any other existing cavities, of a number of teeth and then placing them in a 5% nitrate of silver solution for from 24 to 48 hours. All showed on section a penetration of the silver into the dentin, extending quite deeply toward the pulp. Sections made from pyorrhetic teeth showed abnormally large channels running from the periodontium toward the pulp. The channels stained a dense black, showing the existence of soft tissue. They were found only in pyorrhetic teeth. Interstitial calcification is found in the pyorrhetic pulp and according to Prof. H. Gideon Wells this never takes place in normal tissue. Round cell infiltration is also found in the pyorrhetic pulp and is most likely elicited by bacterial toxins. Fibrosis is the third most frequent lesion seen in the pyorrhetic pulp and represents a scar of a former injury. Pulps in pyorrhetic teeth become involved either via the apical periodontium or through a lateral channel and infection once established within the pulp remains a constant periapical infection. Although the lateral lesions were apparently healed the periapical tissue remained involved until the offending pulp was removed. Pain in pyorrhetic teeth may be traced to a chronic pulpitis. I have never found a pulp removed from a pyorrhetic tooth to be normal. It would appear to be a logical deduction, that where a pathologic pulp is present, a pyorrhetic condition cannot be cured by treatment applied exclusively to the external surface of the root with no treatment of the pulp itself.

—*M. M. B.*

4—Cahn, Lester Richard, D. D. S.—**The report of a case showing the penetration of a stain (nitrate of silver) from the pulp canal to the periodontal membrane and peripheral tissues,** in: Dental Cosmos. lxix: 1164-1171. Nov., 1927.

Lower first molar with acute pulpitis was opened up and as much of the pulp was removed as possible. The canals were then treated and filled with the Howe nitrate of silver, ammoniacal formalin combination. Seven years later a radiograph showed a large radiolucent area at the apical region. The tooth was extracted and the different tissues, soft and hard were prepared for microscopical examination. It was found that the nitrate of silver had penetrated deeply. The stain passed from the pulp canal through the dentinal tubules and cementum to the periodontal membrane, and then even into the alveolar process. It showed that there is a complete communication between the pulp and periodontal membrane and the alveolar process.

—*M. M. B.*

5—Delater, Gustave. **Epulis and pyorrhea usually stages in evolution of an initial latent gingivitis,** in: Revue de Stomatologie xxix (11) 664-685, Nov., 1927.

The authors have previously with Bercher (Val-de-Grace) attempted to simplify recent conceptions as to the origin of granulomas and paradental cysts, of adamantine epitheliomas, epulides, and the majority of the new growths arising from some remains of odontoplastic origin; and to bring together the tumor growths evolving in the peri-apical tissues. Defining their inflammatory nature we have placed them in evolutionary opposition to those disorders ending in suppuration. The author explains how those tumors in the vicinity of the apex progress from a constructive reaction to local septic irritation. While, on the one hand, microbes ordinarily cause destruction, necrosis, suppuration of cells and tissues with which they come in contact—such the violence of their attack—, it may happen that, arrived at the apical extremity of an infected root canal, their vitality and aggressiveness is so weakened, and the integrity of the cells is so slowly compromised thereby, that the neighboring cells find time and means to react and repair the loss of the others. Thus stimulated to reproduction, and overpassing their aim, these cells multiply or secrete to excess, giving rise to diverse new formations; epithelio-granulomas (more or less vacuolated), angiogranulomas, fibroblastomas, etc.—histologic basis for the clinical names, granuloma, cyst, epulis, etc.

In this article the author wishes to establish the same relation between two very different lesions of the neck of the tooth; a constructive process, epulis; a destructive process pyorrhea. Both start from an infection, local and almost constant at a certain age; this may obtain for years without accident except for some intercurrent congestion, but resulting always in the slow

resorption of the alveolar margin. In other cases, it may give rise to sudden outbreaks of destructive and suppurative symptoms (pyorrhea) or to a constructive reaction in the form of a new growth generally benign (epulis).

I. *Latent gingivitis.* The author has noted after careful and prolonged study of tags of gingival tissue, mucous or alveolar taken from patients operated for tumor; or sections of fragments of traumatized jaws in every case on the proximo-dental surface of these fragments of tissue apparently healthy in the adults, signs of an inflammatory condition.

a. The epithelium has lost its layer of flat cells.

2. The cellules of the Malpighian layer show signs of disintegration, cellules separated, protoplasm transparent or vacuolated, nucleus smaller and clearer, and infiltration of polynuclears, showing presence of pathogenic microbes, etc.

c. Architectural modifications of the investing epithelium; interpapillary points seem elongated, arborescent and anastomosed,—characteristic of inflammatory irritation.

d. Below this epithelium, the connective tissue is flabby, myxoid, infiltrated with mononuclear leucocytes, plasmocytes, etc.

All this tends to prove that almost constantly from 15-25 yrs. at the proximo-dental aspect of the gingival margin, there exists an irritation, probably due to a latent gingivitis.

II. *The destructive complication; pyorrhea.* This the author believes to be the latest stage of the latent gingivitis. All the characters pointed out on the apparently healthy gums are to be found on the pyorrheic. He describes the epithelial constructive stage; the stage of inflammatory destruction; and the fibrosis due to the defensive effort of the tissues to protect themselves from further inroads of disease by cicatrisation.

Epulis, the manifestation of the constructive tendency is then described, and the expulsive form of proliferating gingivitis.

The author's conclusions from the observations described in this article are to the effect that his histological studies confirm his earlier reports as to the mechanism by which peridental tumors develop, and the role played by the epithelium. It appears to him a constructive reaction to an attenuated microbic aggression. Since the epithelium is the chief elective focus for the germs of the mouth, it is the ordinary point of departure of the tumor process, whether it starts in an epithelial rest of Malassez, or is seated in the epithelium (embryologically comparable) of the proximo-dental surface of the gingival papilla. In opposition to this evolution, are the destructive processes of suppuration, most often associated, at the apex, with the epitheliogranulomas which mask them; more often at the neck, where they develop into pyorrhea, much more frequently then into epulides. —*H. A. R.*

6—Dewey, Kaethe W., M. D. **Tissue changes and regenerative potency in paradental diseases,** in: J. Am. Dent. A. xiv, pg. 280. 1927.

Author has a predilection for "paradentium" as a collective term to designate all tissue alongside and surrounding the teeth including alveolar process and gingiva and thus these tissues form a biologic and anatomic unit. An increasing number adhere to the theory that predisposing systemic factors play a major part in causation of pyorrhea. Under unfavorable systemic conditions local resistance is lowered and local factors then act as auxiliary and incidental causes. She believes tho that local harmful agencies or conditions alone operating in sufficient quantity and over a long enough period will have a similar effect.

Alterations in the circulation and in tissue tension are the first and also the fundamental factors in the pathogenesis of paradental disturbances and thus constitutional diseases characterized by circulatory disturbances are most important etiologically in inducing the local disease. Of all the pathologic changes in the paradentium in pyorrhea she considers the alveolar bone involvement the most characteristic but the question as to whether this is primary or secondary to the inflammation of gingivae and periodontium remains unsettled although Hopewell-Smith, Talbot, Fleismann and Gottlieb believe that bone alterations are primary.

In considering the osseous change known as halisteresis she discusses at length the

theory of Pommer with regard to new bone formation, in its application to pyorrhea.

Pommer holds that halisteresis is not a simple atrophy or diminution of lime salts, but that following the ordinary bone absorption by osteoclasis or lacunar absorption there is in this condition a new fibrillar bone formation in which there is deficient calcification. Dewey while admitting that vascular changes in pyorrhea may interfere with the formative cells of the paradentium and produce irregular bone—improperly impregnated with calcium salts—thinks we often find simple atrophy and a real halisteresis. However, the resulting bone condition is admittedly the same functionally and anatomically in either case, and underlying causes probably very similar. Author goes at length into the patho-histology of the degenerative bone changes from the time that homogeneous structure is lost in a small localized area in apposition with the periodontium, where it is replaced by fibres running vertical to the surface of the bone, to those extreme conditions involving the entire thickness of the alveolar bone where the decalcified fibres stream out into the mucosal connective tissue. Ultimately these fibres are converted into ordinary connective tissue but retain the original bone outline. Remnants of bone may be found in this transformed tissue.

Hypertrophy of cementum compensates in a measure for loss of bone except where bone absorption is excessive. The increased traction produced by loosened teeth reacts pathologically on alveolar bone and increases absorption and looseness. Epithelial proliferation follows as sequence of vascular changes induced in underlying connective tissue and this proliferation means deepening of the pockets favoring further infection and stimulates formation of granulation tissue. Further epithelial proliferation accompanies the formation of this granulation tissue and thus a vicious cycle is formed. Bone has marvelous regenerative powers and if local toxic and constitutional underlying causes can be removed, more or less complete results may be expected. Interesting experiments with guinea pigs on vitamin-deficient diets and then on corrected diets indicated hopeful possibility of recuperation in badly damaged bony structures.

In the local phase bacteria are an important maintaining factor tho not causative—and improvement observed from the use of germicides, such as spirocheticides for example, is more or less indirect.

The author compares conversion of infected granulation tissue into sterile connective tissue as found in cases of root cysts with the process induced in the treatment of pyorrhea where we encourage hyperaemia and the formation of granulation tissue, and ultimate regeneration. The article is accompanied by excellent photomicrographs; and concludes by emphasizing constitutional factors which should be handled in co-operation with the physician.

—*C. R.*

7—Gottlieb, Bernhard. **Tissue changes in pyorrhea,** in: J. Am. Dent. A. xiv, Dec., 1927.

Author first "lays down morphological and clinical facts" concerning the eruption of the teeth and their relation to supporting structures and sums up his idea of normal "evolution" of the teeth as follows:

1. Migration of the epithelium attachment toward the apex.

2. Simultaneous atrophy of alveolar crest.

3. Protrusion of the teeth toward the occlusal line.

4. Mesial displacement of the teeth.

5. The compensatory abrasion of the teeth both at the grinding surface and at points of contact.

These processes are continuous though at varying pace throughout life from time when tooth breaks through united mouth and enamel epithelium leaving the entire outer enamel epithelium joined to enamel surface as before.

Bottom of pocket (base of gingival crevice) stays no longer at cemento-enamel junction than anywhere else. Eruption of teeth continues instead of stopping here as generally supposed, rate only becoming slower; and later on rate may become abnormally rapid again, great variations occurring because of systemic factors, such as rickets which greatly retards eruption. but these variations are merely different phases of a continuous biologic phenomenon.

True hornified cuticula dentis is formed of outer enamel epithelium. It covers over

cementum also as far as the downward-grown epithelium extends and is very resistant to alkalies and acids and may stop entrance to enamal lamellae. Downward growth of epithelium commences before eruption reaches cemento-enamel junction. Normal crest of process has fixed relation (within physiologic limits) to bottom of pocket and this maintains as long as eruption of tooth, extension of epithelial attachment and absorption of crest proceed concurrently. There is no sharp distinction between this, which is physiologic, and the pathologic. Depth of pockets varies from nothing to one or more millimeters without crossing line to the pathologic, and same variation exists within the normal, for length of epithelial attachment.

To be within physiologic limits eruption rate should be slow enough so that part of tooth remaining unerupted in jaw is at all times sufficient to support mastication. Also abrasion of the chewing surfaces should keep pace with downward growth of epithelium to prevent extra-alveolar lever from becoming abnormally long; for excessive leverage of elongated clinical crowns damages suspensory apparatus even under normal use, normal function here becoming traumatic.

When accelerated epithelial migration takes place approximally with neighboring parts in situ and gingival margin cannot follow pari passu, the deepened pocket becomes subject to inflammation and infection. Also gingivitis being a hypertrophy may cause failure of gingival margin to follow deepening pocket. Even where a deepened pocket is "normal" inflammation may result from this morphology, and in both cases therapeutic intervention must be resorted to.

Gottlieb finds the presence of a pocket the chief requisite for existence of pyorrhea, inflammation and bacterial activity being secondary.

Before downward growth of epithelium can proceed, the connective tissue fibres must become detached from their anchorage in cementum from trauma or inflammation; Epithelium then proliferates to cover exposed surface. Detachment and proliferation takes place on side opposite application of strain where fibres are relaxed. Inflammation which destroys connective tissue fibres must abate before epithelium can proliferate, and again increase to destroy more fibres. Connective tissue for unknown reason does not reorganize or reattach.

Cementum is not dead merely because separated from connective tissue attachments. All cells are subject to law of molting and cementum cells perish also and the fibres imbedded lose their hold unless new cementum has been deposited to give reattachment. Thus normally a tooth requires continuous deposition of new cementum to maintain it in function. When normal functional demands are exceeded (traumatic occlusion) a resorption of root or alveolus or both occurs with a widening of periodontal membrane, atrophy of suspensory fibres and loosening of tooth. When a bundle of fibres loses its hold the functional stimulus to the alveloar bone is also destroyed; all this damage, can be entirely repaired starting with new cementum, then insertion of fibres and, with re-establishment of functional stimulus, reconstruction of alveolar bone. Functional demand is always necessary but as long as surface of resorption is not covered by epithelium it can be repaired by cementum as can also alveolar bone opposite resorbed area. If however cementum fails to redeposit, enlarged periodontal space persists with all its injurious sequellae.

We see that cementum formation is of first importance.

Gottlieb distinguishes between marginal and diffuse atrophy—the first due to neglect with inflammation and suppuration as early symptoms, and loosening follows later. Diffuse artophy is accompanied by what he calls "paradental" pyorrhea. Wandering and loosening come first here with pocket formation and suppuration later.

The author thinks long rooted teeth most to be desired and to this end we should try to develop large type of man free from tendency to rickets and strive to discover a way to influence the rate of eruption of the teeth. Lastly and perhaps most important we should seek to discover whether the continuous deposition of cementum on root is controlled from any center of the organism which our prophylactic measures might reach and influence in a favorable direction, or whether we might accomplish this by theraupeutic measures.

This fine scientific article has practical

value and is illustrated with many excellent photomicrographs. —*C. R.*

8—Gottlieb, Bernhard. **Paradentalpyorrhoe und Alveolaratrophie,** in: Fortschritte der Zahnheilkunde, May, 1927.

The clinical expression of alveolar atrophy is, as is well known, loosening of the teeth. The necessity of constant control of the articulation, remembering that the teeth practically never remain through life in ideal articulation has been discussed in earlier papers.

In spite of theoretical protests from many quarters, the teeth continue to erupt through life with varying degree of rapidity. It is impossible for them to remain in their ideal occlusion if their sideward movement is not continually renewed. Our task is to control this sidewise movement when it occurs. This control is accomplished most ideally when one of the articulating surfaces is metallic. Gottlieb then discusses the construction of prostheses.

Gottlieb says that Mayer reports that he has found, in heavy cement deposits in the deeper layers, empty bone lacunae, while in the newer layers the bone corpuscles were well preserved. In these "disturbances of the cement vitality" Euler and Mayer see "chiefly a kind of Vorstadium—preliminary stage of atrophy." From this we must conclude that these deep lying parts "with disturbed vitality" would fall apart, unless new layers of cement were laid down. Gottlieb himself showed (Fortschritte d. Z., 232, 1925) a microphotograph of pig cementum with empty bone lacunae in the deeper portions and the well preserved bone corpuscles at the surface. He then pointed out that the pig cementum was suitable for this demonstration, since in transition and especially in the milk tooth stage as well as in the permanent dentition, only bone cement is present.

Gottlieb calls attention to the work of Euler and Mayer under the title *"Parodontitis chronica marginalis progressiva"* (Alveolarpyorrhoe). A symptom which is often the first to draw the patient's attention to his jaw troubles "is the increased spacing between the front teeth and definite changes in the position of other teeth." This is the "wandering" which G. holds as characteristic of diffuse atrophy. Gottlieb does not consider this an early symptom of parodontitis marginalis. There may be no trace of inflammation of the marginal portion of the paradentium whatever, while in spite of this a pathologic condition is present.

It is worthy of note that Euler and Mayer described every epithelial proliferation along the cementum as "abnormal." In this sense every denture is abnormal, since there can scarcely be found the denture of a full-grown man (about 30 yrs.) whose teeth show at no point an epithelial proliferation along the cementum. This standpoint (of Euler and Mayer) shared by Haüpl, has grown out of the fact that neither in milk or permanent dentition is a deep proliferation ever observed along the cementum embracing simultaneously the entire circumference of the tooth. In Gottlieb's own histologic sections in which on technical grounds we cannot as a rule see the entire circumference at once we never see the deepest point of the epithelium override the amelo-cemental boundary at all points at the same time. It is then unlikely, that, almost without exception, each tooth at one time in life is normal on one side and pathologic on the other.

Chaim writes: "The *processus alveolaris,* weakened in its resistance by Ca deficiency yields under the pressure of the altered volume of the gingival papillae."

Gottlieb does not follow him here. (a) Resorption of the alveolar process in consequence of the pressure of the swollen papillae is clearly not thinkable, since the papillae except at their bases are free on all sides; and any tension present will scarcely be directed against the alveolar margin. As little is the point clear that the processus alveolaris is weakened by lack of Ca. It is characteristic of alveolar pyorrhea that nowhere else in the bony system are disturbances manifested, and one can hardly think that lack of Ca in the blood would work itself out only through the alveolar process. No one has yet discovered symptoms of Ca poverty in the alveolar atrophy. Let Chaim administer endocrine preparations (e. g., testogan, ovarialopton) says Gottlieb, and report again.

Gottlieb appears to agree with Siegmund and Weber that "in principle the like relations serve for the appearance of resorption in the dental organ without primary pathological changes in the soft tissues—for the

acutely and chronically inflamed parodontium and for the resorption in impacted teeth."

Physiological resorption of the roots of the milk teeth is the result of heightened intermediary metabolism in the investment of the substitute germ, which serves to ward the upbuilding of the latter. All resorptive processes are, according to the histologic changes to be thought of as resorptive inflammations. The specific resorption tissues are "the product of metabolic changes which take place either on the basis of local inflammatory processes and changes in the occlusal stress; or as local appearances of constitutionally conditioned lessened value of the hard tissues. The histologic changes in the soft tissues are in these cases the same."

Weski, who makes the chronic marginal inflammations of the alveolar bone responsible for this (i. e., the degeneration and upbuilding of the surrounding tissues) puts a horizontal and a vertical atrophy in place of the marginal atrophy of Gottlieb—that form which spreads along the long axis of the tooth, making a deep V-shaped pocket from the alveolar margin upward. In the horizontal form, which runs parallel to the gingival margin, the entire alveolar margin is resorbed."

In the marginal atrophy which is related to gingival marginal injuries, the alveolar margin disappears, while the width of the periodontal space remains the same in the uppermost layers of the tooth surface. Gottlieb distinguishes yet another form—a diffuse atrophy in which the alveolar margin disappears irregularly and the teeth begin to wander.

Finally the desquamation of epithelium plays an important role. —*H. A. R.*

9—Haupl, C. Ueber traumatisch verursachte Gewebsveränderungen in Paradentium, in: Zeitschrift f. Stom. xxv (4) 307. 1927. Abs. Fortschr. d. Z. iv (2) 507-508. 1928.

Paradentium according to Weski, is the physiologic functional unity of gingivae, alveolar bone, periosteum, and tooth (otherwise called the tooth investing or supporting tissues). Next to the functional unity stands the anatomic unity.

The paradental soft tissue (gingiva) consists of an epithelial and a mesodermal portion. The tiny crevice in the duplicature of the epithelium at the apical third of the tooth is called the gingival pocket. The mesodermal portion consists of the subepithelial and supra-alveolar connective tissue; the former overlies the cemento-enamel junction, the second (the circular ligament) lies between this and the alveolar bone. The paradental bone consists of the alveolar bony process which is in connection with the adjacent investing tissues and the body of the jaw bone. Between the bony lamina and the cement of the tooth lies the periosteum (dental). The function of the supporting tissues consists in resistance to the mechanical stresses affecting the tooth. The vessels with their incompressible fluid contents act as a sort of spring.

The inflammatory alteration of the paradentium, paradentitis, is divided into (1) marginal and (2) apical. Haüpl is dealing with the former in this article. As a result of the physiologic unity, changes in the superficial parts of the paradentium bring about similar changes in the deep parts. The epithelial changes are partly regressive, partly progressive. In the former case, the epithelial cells show necrotic changes, going on to loss of epithelium and ulceration reaching to the floor of the pocket. The epithelium loosened by edema is infiltrated by leucocytes. The progressive processes manifest themselves in epithelial proliferations especially along the cement. The tension of the subepithelial and also the supra-alveolar connective tissue undergoes a change; then follows the increased growth of the deep epithelium owing to the heightened nourishment of the cells of a tissue loosened by an inflammatory edema; and through the stimulation (nekrohormonal).

The points of attachment of the fibrous bundles of the supra-alveolar connective tissue to the cementum seem to be points of lessened resistance, through which pathologic loosenings, bacteria, etc., easily enter, thus favoring disease processes. The loose connection of the deep proliferating epithelium with the cement substance gives occasion for the formation of the pathologic pocket. In it collect various materials which stimulate the inflammatory processes; e. g., desquamated epithelial cells, food debris, calculus, and above all bacteria, whereby the development of the inflammatory process is

furthered. Beside the inflammation there is also a histiocytic reaction, that is, an ab- and re-sorption.

Next comes a loosening of the paradental soft tissues and a weakening of the supporting structures which also takes effect upon the apical portions of the tooth. In the bone also is formed a pocket by osseous degeneration, also a regeneration of bone occurs, however, chiefly in young individuals. In atrophic disturbances the osteoblasts are often no longer in condition to form a tissue so highly specialized as true bone, so that they form only connective tissue.

These processes in bone are osteitis, and osteomyelitis, chronic in character. The first degenerative change in the paradentium is a hypertrophic thickening of the cement, corresponding to the degenerative process going on in the overlying bone. Through these heightened processes of destructive metabolism as a result comes irritation of the vascular system leading sometimes to formation of granulation tissue.

Haüpl distinguishes predisposing and intrinsic causes: (a) physiologic organic diathesis, besides tooth deposits, tartar, overhanging fillings, etc., incorrectly designed dental work, clasps, etc. Intrinsic causes are mechanical, traumatic, chemical, toxic, infectious (pathogenic micro-organisms).

The clinical symptoms of marginal paradentitis are sometimes concealed under the appearances of gingivitis to which, beside the pocket, the denudation of the tooth surface points.

In treatment the gingivitis is first to be overcome; the aim to be sought is a clinically sound gum that is firmly attached to the tooth surface, and a firmly fixed tooth. Gingivectomy is indicated by pocket depth of over 3 mm. The radical surgical method is called for in bone pockets and cavities. H. further discusses use of splints, chain, etc.; gold inlays with pivots; finally, if necessary, extraction. Transl. from Abstr. in Fortschritte. —*H. A. R.*

10—James, W. Warwick, and Counsell, Arthur. **Histological investigation into "so-called pyorrhea alveolaris,"** in: Brit. Dent. Jour. xlviii: 1237-1252, Oct., 1927.

After summarizing their findings on the normal structures and pathological changes in pyorrhea, the authors give their conclusions as follows:

"Our work has been concerned with the primary changes occurring in 'so-called pyorrhea alveolaris.' We have not discussed lesion resulting from secondary bacterial infections.

"We believe that we have produced sufficient evidence to show that the primary lesion is associated with the presence of deposits on the tooth.

"Of the supporting structures of the teeth, the first tissue to be affected is the subgingival epithelium.

"The path of invasion passes through the gum corium to the alveolar bone, involves the bone marrow and later the periodontal membrane.

"Progressive destruction and reformation of tissues occur. A pocket results which is constantly lined by epithelium.

"The presence of pus is unusual and when present is due to secondary infection, pyogenic organisms such as staphylococci being readily demonstrated in the pus. The periodontal membrane although replaced in its upper part by new formations, is the last tissue to be involved, via the bone, in the process of infiltration. Our work supports the view that the primary lesion is of local origin. We are in agreement with much that has been said by Znamensky and Goodrich and Moseley. We have found the exhaustive work by Gottlieb and his co-workers most helpful, but we cannot support his hypothesis that many of the changes found are due to a physiological continuous eruption." —*H. J. L.*

James, W. W. and A. Counsell. Br. Dent. J. Oct., 1927. **Editorial note, same issue,** p. 1270. D. Cosmos, Jan. 1928-117.

There should be no necessity to draw attention to the importance of this paper. The careful and methodically conducted investigation recorded throws a valuable light upon the processes taking place in the production of this very common but little understood condition, and although they leave important problems still unsolved, they are confirmatory of explanations from frequently insufficient data given by some observers, and tend to disprove other conclusions drawn from similarly incomplete evidence. Although the actual period at which normal

processes pass into an abnormal stage has not been by any means established, the earliest beginnings appear to be always associated with calcareous deposits in which a basis of leptothrix buccalis can usually be demonstrated, as was suggested by the experiments of Arthur Bulleid. If, as the authors state, the subgingival epithelium, the gum corium, the alveolar bone and the bone-marrow are affected in the order named, and last of all the periodontal membrane is attacked, some of the hypotheses upon which the present more or less empirical methods of treatment have been based will have to be reconsidered. The whole question is one of the greatest possible interest, and Messrs. James and Counsell are to be congratulated upon one of the most important attempts at its solution. —*Copy.*

This paper is well illustrated with photomicrographs and should be carefully read by all students of periodontology. The work here reported ranks well with that of Gottlieb in Vienna and Box in Canada.—*H. J. L.*

11—Johnson, A. Leroy, with Appleton, and Rittershofer. **Tissue changes involved in tooth movement,** in: Int. J. Orthod. and Or. Surg., Oct., 1926. Abs. Cosmos lxix: 219-220, Feb., 1927.

The work was done on a monkey, Macacus rhesus. A lingual arch was used with an auxiliary spring. After the movement was grossly recognizable the animals were killed and sections made after embedding in celloidin, and stained with haematoxylin and eosin. The authors observations were as follows:

1. There is a deformation at the apical end of the tooth that has been moved.

2. Changes in alveolar structure.

a. Resorption of labial osseous tissue in the apical region as indicated by an indistinctness of definition between the alveolar bone and the alveolar periosteal membrane.

b. The direction of trabecular structure conforms to the direction of the tooth movement.

c. The relative number of osteoclasts greater on the labial side of the apical region of the experimental animal than in like region of the control.

3. Resorption area in the deformed section of the lingual apical region of the 40 days experimental teeth. —*Copy.*

12—Köhler, Josef. **Verwachsung zwischen Zahn und Knochen. Fusion between tooth and bone,** in: Ztschr. f. stomat. xxv (9) 896, 1927.

The jaws and teeth of two old dogs were examined. Macroscopically the teeth of No. 1 (10 yrs. old) were mostly anchored fast in the jaw; many were deeply abraded; the gingival margins were inflamed and the teeth covered with tartar; while the teeth of dog 2, also 10 yrs. old, were loose and ready to fall out. The marginal gingiva was inflamed and the teeth heavily coated with tartar. Histologically, dog 1, showed 14 of 28 teeth grown together with the alveolar bone; the other teeth showed marked cement hyperplasia and epithelial proliferation. 5 out of 19 teeth of dog 2, were fused with the bone; the findings in dog 2 were directly the opposite of those in dog 1; deep pocket formation, marked evidence of resorption, paradental abscesses. Concrements were found irregularly on all teeth with exception of the bifurcations of molars and premolars. *Transl. Abs.*—*H. A. R.*

13—Kotanyi, E. **The changes of the teeth and surrounding tissues in experimental scurvy of the guinea-pig,** in: Zeitschr. f. Stomatologie, July, 1927. Abs. D. Cosmos lxix: 1077, October, 1927.

Enamel. Concerning the alterations of the enamel as shown by decalcified sections stained with hematoxylin, we have come to the following conclusions: we must distinguish two types; in the first we find the degeneration of the ameloblasts in the foreground, in the second the alteration of the enamel. To the first group we assign those cases in which the structure of the enamel is intact; to the other group those in which the enamel is split up into fibers where the ameloblasts are normal.

Dentin. Concerning the alterations of the dentin as shown by decalcified sections stained with hematoxylin, we have come to the following conclusions: the first alterations of the dentin exist in the widening of the dentinal tubes. The form of the dentinal tubes in cross-section is variable; most of the fibers of Tomes have degenerated. The signs of the greatest degeneration of the dentin are cysts. We find very often the altered parts of the dentin elliptical; their limits are more deeply stained with hematoxylin than surrounding tissues, because

probably the dissolved calcium is precipitated here. We find these alterations of the dentin in four-fifths of the cases when the animals were nourished in winter; when, on the other hand, the animals were fed in summer, the same alterations were shown only in about one-tenth of the cases.

Calcification of the dentin. The degree of calcification of the dentin and the relative thickness of dentin and enamel are dependent on the diet of the animals.

Central ends of the teeth. In the cases where the animals were fed on a diet with only a small amount of vitamin C, the central ends of the teeth were mostly normal. In other cases, where the animals were fed for a very long time on a diet devoid of Vitamin C and then for two weeks on the same diet with the addition of lemon juice, we find the central end irregular and the formation of enamel and dentin disturbed. The third group of figures shows infractures and fractures. These animals were fed upon a diet partly deficient and partly devoid of Vitamin C.

Cystic degenerations. We find in experimental scorbutus cystic degenerations in all the tissues in and surrounding the tooth.

Pulp chamber. In the pulp chamber we find all forms of degeneration of the connective tissue from fibrous degeneration to pulp bone formation. The odontoblasts are destroyed very soon after the beginning of a diet devoid of Vitamin C. We find a very different structure of the pulp bone and in many cases osteodentin. The alterations of the connective tissue of the pulp chamber consist (1) of very varying forms of degeneration; fibrous degeneration, bone and osteodentin formation, etc.

(2) Of the formation of cysts; and

(3) Of hemorrhages.

The physiological variation in scurvy is very great. From a given stained section, it is impossible to determine quantitatively the amount of Vitamin C in the diet.

—*Copy*

14—Kronfeld, Rudolph. **Spielt die Qualit der Hartsubstanzen bei der Resorption eine Rolle,** in: Zeitschr. f. Stomat. xxv (11) 1099-1109. 1927. Abs. Dtsche. Mschr. d. Zahnheilk. 1928.

The author describes his investigations as to whether under normal physiological condition, the calcium content of the tissues plays a part in the resistance of the tissues to resorptive processes. He finds an isolated lack of calcification ("Stehenbleiben") of Ca free cementum and dentin in roots already far resorbed; and comes to the same conclusion of the dependence of the process of resorption on the Ca contents of the tissues as Pommer has already presented in studies of rickets and osteomalacia.

Transl. abs.—*H. A. R.*

15—Lewis, J. M., D. D. S. **Some observations on the pathology of pyorrhea gingivae,** in: Australian Dent. Jour. xxxi (12), Dec., 1927. Abst. Brit. Dent. Jour. xlix (4), 175-177, Feb., 1928.

This name is substituted for pyorrhea alveolaris as it emphasizes the fact that the chief source of pus is not the alveolus or the periodontal membrane but the soft tissues. Its use is not limited to one particular phase of the disease, nor does it exclude those cases in which absorption has deprived the tooth of its support and created an abnormally deep pocket. Marginal gingivitis is easily induced by the lodgment of decomposing carbohydrate food at the neck of the tooth; and in the course of a few days in some individuals will produce a hyperemic area which bleeds easily and may even be denuded of epithelium. To such beginnings a large proportion of well established pyorrhea may be traced. The author then describes the varying modes of onset and symptoms of pyorrhea with the usual discussion of the histopathological appearances, as described by Gottlieb, Box, Stillman, and others. He thinks the "intermediary substance" is strictly confined to the portion of the tooth covered by enamel and does not appear to be continuous with the "tooth cuticle" described by Gottlieb. In a typical section the alveolar tissue may show the following features. The medullary spaces are occupied by a delicate mesh-work supporting irregular thin-walled vessels. In some areas the medullary stroma is dense; in others fibrous tissue is mixed with the more delicate stroma. Many of the larger spaces show islets of alveolus undergoing absorption. Collections of nuclei suggest the presence of giant cells. Mononuclear cells are scattered in the stroma.

Alveolus which has reached this condi-

tion has unsuspected powers of recovery, and in many cases will become recalcified after the removal of the local factor producing the atrophy. But whatever regenerative changes may be brought about an actual addition to the alveolar margin must not be looked for even in the denser interdental septa. —*Copy of abstr.*

16—Rywkind, A. W. **Die Bedeutung des Deckepithels der Mundhöhle für die Morphologie und Pathologie der parodontalen produktiven Vorgänge,** in: Dtsche Mschr, f. Z. Heft 15, 1927. Abs. Ztschr. f. Stomat. xxvi: 656, 1928.

Summary and conclusions. In most cases, the microscopic investigation of pathologic proliferations in the parodontium yield consistent morphological appearances. The morphologic similiarities are the presence of formed epithelium in the connective tissue, and the typical arrangement of the epithelium after the type of the Malpighian layer of the oral epithelial covering.

The form and arrangement of the epithelial tissue in the pathological parodontal proliferations corresponds in general to the morphology of the epithelium in the period of odontogenesis. So far as it is possible to determine on the basis of the morphologic picture, the proliferations of the epithelial covering of the mouth in the parodontium develop along the same line as is peculiar to them in the process of odontogenesis. The pathology and clinical appearances of parodontal pathologic processes is in close relation to the evidences of this developmental trend—the sinking in of the epithelium in the underlying tissue, the loosening of the epithelial tissue with formation of stellate cells, and later the separation of the epithelial trabeculae. —*Copy*

17—Sassier, Pierre. **Lymphatics of the gums,** in: Annales d'anat. pathol., IV: 212-215. Feb., 1927.

These lymphatics have been studied on each one of 50 subjects. They enter always the submaxillary ganglions, often the submental ganglions; and accessorily to the ganglions, the inferior parotid, the inferior genial, the anterior jugular digastric. The lymphatic channels and the ganglionic terrains are variable for each jaw.

In the maxilla the injections were pushed to the apex of the incisors, canines, molars or premolars, to ascertain whether there existed for each of these gingival areas a ganglionic area. This was not found, for actually all the lymphatics pass to the submaxillary ganglion exclusively.

At the point of injection the authors found a fine mucous network which gives off three or four lymphatic trunks passing first to the cheek, and gaining finally the facial vein.

These gingival lymphatics have received many names. Author classifies them as follows:

1. Preglandular—in front of the submaxillary gland.

2. Pre- or retrovascular—in front of or behind the facial vessels.

3. Behind the gland—retroglandular.

4. Intracapsular—within the gland capsule.

There is only one preglandular ganglion; most often one retrovascular, sometimes two. This author in spite of his extended researches has never met the intracapsular g.

The arrangement in the mandible is a little more complex. Author describes the main lymphatic paths and various accessory paths. These vessels may follow the same paths as the others; or after perforating the mucosa they may finally penetrate one of the ganglia of the internal jugular chain.

The search for this ganglion is most important because in case of neoplasms of the gingiva, this ganglion may be reached by direct extension.

Summary:

1. The ganglions for the upper gingiva are always submaxillary.

2. For the lower jaw.

a. For the mandible—always submaxillary—1 in 12, inferior genial.

b. For the incisor area—always submaxillary—1/3 of cases, submental.

c. For the molar area—always submaxillary—8 in 100 cases, inferior parotid; 8 in 100 cases, substernomaxillary. —*H. A. R.*

18—Simonton, F. V., D. D. S. **Parodontoclasia,** in: Pac. D. Gaz. xxxv (4), 251-265. Apr., 1927.

The microscopic study of the investing tissues of the teeth reveals three phenomena

of major importance in relation to parodontoclasia.

1. Inflammation.

2. Involution of epithelium along the surface of the root.

3. Resorption of the alveolar process.

Prevailing opinion in this country holds that the inflammation is the cause of the other two conditions; yet recent findings, notably those of Gottlieb, raise a question whether these three factors may not be distinct and arise from quite different causes.

The author gives a formula for the expression of retrograde changes caused by local or systemic agencies or both:

$$\text{Retrograde changes in parodontium} = \frac{\text{Local exciting factors}}{\text{Resistance}}$$

Destructive changes will be augmented either by increase in strength of local factors or by decrease in the resistance.

These changes may also occur through endogenous causes without presence of local factors.

Author gives a more recent classification: Inflammatory type—Parodontitis (Chemobacterial (Schmutzpyorrhöe)).

Parodontosis—systemic.

In the description of both of these types author follows the lines laid down by Gottlieb; although he cannot quite accept Gottlieb's view that "filth pyorrhea" is not a disease, and the establishment of hygiene cannot be regarded as a therapeutic measure.

The dehiscence of the cuticula dentis and the downgrowth of epithelium is then discussed. Author believes that much of Gottlieb's theory is well established.

He summarizes thus:

1. Chemo-bacterial parodontitis (filth pyorrhea) appears to be largely dependent upon two important factors: 1. The presence in the mouth of debris from artificial and artificially prepared food (easily decomposed); 2. Some systemic factor which controls the hygiene of the mouth.

2. Recent evidence suggests that the etiologic factor in systemic parodontitis (diffuse atrophy) may be a disturbance of the acid-base balance of the diet.

3. Systemic parodontitis. Histologically this condition seems to be a sequel to gingivitis. Cause is at present unknown.

4. Dehiscence of the cuticula dentis and downgrowth of epithelium along the root, as has been shown by Gottlieb, are phenomena of great importance and demand much further study.

19—Skillen, W. G., and Emil Mueller. **Epithelium and the physiologic pocket,** in: J. Am. Dent. A. xiv, 1149, 1927.

The authors state that this paper is limited to a review of "the newer conception regarding tooth eruption, the cuticula and physiologic pocket development" at the same time that they offer corroborating evidence attending their own researches on pigs, dogs and sheep. Considerable study is given to Nasmyth's membrane, sometimes referred to as enamel cuticle, cuticula dentis, etc., and the statement made that "it is intimately involved in all diseases of the gums, particularly pyorrhea, and probably plays an important part in the etiology of caries." Comparison is made between what the authors call the physiologic and pathologic pocket, referring to what is usually known as the gingival crevice and express the opinion that "under physiologically ideal conditions, we believe the pocket depth of 0 mm. is the ideal. The ordinary functioning of the mouth, in the vast majority of instances, brings about a change from this ideal so that a pocket is developed," which must therefore be regarded as the physiologic normal. This it is believed, is developed as the tooth erupts, by separation between the enamel epithelium and Nasmyth's membrane. The article is illustrated with a number of photomicrographs of material used by the authors in their investigations.

—*A. H. M.*

20—Thomas, Newton G., M. A., D. D. S. **Elastic fibres in peridental membrane and pulp,** in: Jour. Dent. Research, VII, 325-326. 1927.

Peridental membrane is single layered and is genetic of calcified tissues on its two surfaces. Up to the present time the statements of Kolliker in 1867, Black in 1887 and Scheff in 1924 that the peridental membrane contained no elastic fibres have been accepted as correct. Using Weigert's resorcin fuchsin as a stain the jaws of a dog were employed which showed elastic fibres abundantly scattered throughout the peridental membranes of all the teeth. They were fine, and arranged usually in the plane of the

white fibres, which they sometimes accompanied but they also showed absence of arrangement in places. The adventitiae of the larger pulpal vessels also contained elastic fibres. —*M. M. B.*

21—Thomas, Newton G., M. A., D. D. S. **Significance of absorption of teeth and alveolar process,** in: J. Am. Dent. A., XIV—2165. 1927.

Author summarizes his paper as follows: "Osteoclasts as agents of absorption are still debated by cytologists, although they are always found in developing bone or areas of bone change as well as in contact with deciduous teeth during the successional period. The periosteum as a factor in bone development and change is similarly discussed. The etiology of tooth loss in the second dentition is unknown. Local irritation from malocclusions either from natural or artificial origins; gingival and apical irritants, as well as chemical or thermal effects, may well be accredited as causes of both internal and external absorption, altho others that escape recognition assuredly occur."

However there is much detail given as to nature of osteoclasts as well as osteoblasts, and also part played by periosteum in bone building. Some twenty authorities are quoted on the genesis and the role of osteoclasts alone in physiological and pathological bone and tooth change, the extensive bibliography being in itself most interesting.

Osteoclasts and osteoblasts are physiologic concomitants but in the case of absorption of temporary teeth, "the cycles of osteoclastic activity" are too rapid to allow reconstruction. Once begun the loss of the teeth is seldom retarded under normal conditions but the stimulus to this activity is unknown since it occurs even when no succeeding tooth is present. Osteoclasis is not the only method of bone resorption or removal. Bone is frequently removed without recognizable cause—probably the result of some vague neurotrophic disturbance.

Thomas refers to the rapid loss of implanted and replanted teeth and implies his condemnation of the practice.

Wells thinks there is no difference in the process of normal and pathologic calcifications and that these as well as decalcifications depend on physico-chemical rather than on chemical processes.

Thomas deprecates the failure of the profession to arrange and make available the vast amount of clinical information stored in files of dental schools and elsewhere. "Causes of bone and tooth absorption must be known before hope may be nurtured for inhibition and cure." —*C. R.*

22—Turner, J. G. **Histology of the periodontal sulcus,** in: Brit. Med. Jour. I: 998-999, 1927; also in Amer. D. Surg. xlvii: 519-521, 1927.

After describing the intimate structure of the region the author continues: "Thus speaking technically, there is a breach; practically there is no breach till the enamel is worn thin by caries or attrition or pyorrhea has disrupted the epithelial attachment to the teeth." In the author's opinion the periodontal space is not always present; if due to develop later it must be provided by the epithelium. In most cases, however, it seems to have been provided for from the beginning. At the time of eruption there is an epithelial lining to the tooth crypt from the outermost epithelium of the enamel organ. This must either blend with the gum or atrophy. The author thinks that it generally atrophies.

Does the gum epithelium attach itself to the tooth; or is there normally a breach of epithelial continuity? In many cases the gum is attached directly to the tooth crown. Some cases show no sulcus possible; some show a shallow sulcus, not at the neck but farther along the crown; some a spot, rather than an actual sulcus. The gum epithelium may spread over the whole crown or may be continuous with Nasmyth's membrane only at the edges, or only at the neck, i. e., attached to the cementum. —*H. A. R.*

23—Wolpe, Paul. **Ueber Verhornungserscheinungen am Zahnfleischepithel. Concerning evidence of cornification in the epithelium of the oral mucosa,** in: Deutsche Mschr. f. Zahnheilk—xlv: 1927.

Widely differing opinions as to this subject are found in the literature. In some textbooks of normal anatomy it is not described at all; in textbooks of pathological anatomy evidences of cornification are mentioned only in regard to squamous-celled

carcinomata, in hairy tongue (lingua nigra) and leukoplakia.

In studies of difficult dentition not yet completed, and its entire histologic relations, as the cause of which we must recognize the deep persistent gum pockets, the author's attention was drawn to the horny appearance of the upper layers of the epithelium at the site of the lower wisdom tooth.

Summary: The author studied, in all, 32 gum sections prepared for histological study, 12 from paradentoses, and 13 from the cadaver. Besides Ehrlich and Van Gieson stains, the Gram stain was used as specific for keratin. In almost all specimens a cornification was present, partly hyper—partly parakeratosic. Cornification is to be regarded as a metaplasia of the keratin-free epithelial gum margin.

In our material there is a close correlation between cornified epithelium and round cell infiltration in the tunica propria,, which shows that in marked cornification the infiltration is slight and conversely, in case of marked infiltration the cornification reaches only a low grade.

The cornification of the epithelium like the round cell infiltration in the tunica propria may be regarded as a defensive reaction of the organism against irritation from without.

—*H. A. R.*

24—Zilkens, Karl. **Pathologische Histologie. Veränderungen der Pulpa bei Paradentose,** in: Fortschritte der Zahnheilk. 1927, p. 284.

Author cites 25 cases described by Craney (Correspondenzbl. f. Zahnärzte il S. 317, 1925) "Die pathologisch-anatomischen Veränderungen der Pulpa bei Pyorrhea alveolaris," (article not obtainable) from all strata of population. He used only caries free teeth, and previously made X-ray pictures to use the bone pictures as a control for the grade of disease. He distinguished following grades:

1. When a third of the alveolar process was absorbed.

2. When atrophy had advanced up to the half of the tooth.

3. When more than half the bone or the entire alveolar margin was gone so that the tooth rested only in granulation tissue.

The pathological changes consist chiefly in a more or less severe atrophy of the pulpal deposit of secondary dentin, hypertrophy of the odontoblast layer and hyperemia of the entire pulp. With increased grade of severity the changes in the odontoblast layer and secondary dentin were increased. Craney reaches the conclusion that the pulp changes are not responsible for the parodontosis; since in all three groups both light and serious changes were found. Teeth in the same mouth showing clinically wholly different grades of parodontosis were histologically very similar—although with the increased degree of parodontosis, the pulp symptoms also increased in severity. Craney thinks that perhaps exogenous stimuli reach the pulp. The blood vascular changes, he, with Römer, looks upon as chronic congestion, the result of inflammation in the socket.

For the practitioner the question of devitalization becomes important. Craney advances the following points against devitalization:

1. In the worst cases clinically, a well developed pulp may be present, disturbance of which would be superfluous.

2. As a result of the usual denticle formation the root treatment is very difficult; while in most cases the pulp cannot be radically removed.

3. When it is proved that the pulp has no influence on the development and persistence of the parodontosis, why should the patient be tormented without necessity?

Praeger, says Zilkens, recently investigated, in a case of sepsis in a woman of 47 years, 26 teeth affected with parodontoses; the loosened teeth with their suppurating pockets were removed. Histologic examination showed secondary dentin formation in the pulp with numerous fixed and free denticles on caries-free teeth. In the pulp itself there was an atrophy of specific pulp cells, increase of intercellular substance and dilatation of the blood vessels. In some sections, the pulps were still rich in cells with retained odontoblast layer. Especially in the most affected molars were found thickenings at the apices and bifurcations. The cement layer extended toward the root; and palatinally and distally was more marked than mesially or buccally. Structurally, Praeger distinguishes two varieties of cementum—one with Sharpey's fibers, and another with

very little structure, resembling alveolar bone; as well as all stages of both kinds.

Thus Zilkens concludes, in spite of severe cement hyperplasia, a high degree of parodontosis may exist. —*H. A. R.*

1928

25—Adloff, Dr. P. **Zur Frage der reaktionsfähigen und wenig reaktionsfähigen Gebisse im Sinne Gottliebs,** in: Vrtlsschr f. Z. xliv (3) 490, 1928.

Adloff says that the Gottlieb School has gone to much trouble in order to base its theories on a broad foundation by bringing Comparative Anatomy to the support of their manifold hypotheses and speculations. This is certainly praiseworthy, he says, but, considering man's wholly unnatural diet it is very difficult to compare his teeth with those of mammals.

He expresses himself very fully as to a theory which seems to him especially important as it plays a very special role among Gottlieb's theories. It is the claim of a special vitality of the cement and the division of the dentition into a part capable of reaction and a part less capable of reaction, etc., since in the first case by deposition of cement on the root disintegration of the bone is compensated, and thereby the tooth continues to remain fast in its socket; while in the other case only weak cement is present, and thus the tooth is unprotected from gradual loosening. Kronfeld, Gottlieb's disciple, has recently reopened this discussion; and does not limit his investigations to human teeth, where this antithesis has a certain significance, but goes into Comparative Anatomy and cites the herbivorous animals as typical examples of the reaction-capable dentition as opposed to the type of man and of beasts of prey, representing the weak reaction capacity. In the herbivora, thinks Kronfeld, the teeth have a higher vital significance. These animals must take their food in large quantities and crush and grind it very thoroughly in order to reach the assimilable substances contained in it. Their denture must then be very resistant and this necessity finds its morphological expression in massive cement which covers even the crowns of the teeth, even over the enamel covered portion. "A free living herbivore without teeth must starve." On the contrary, for men and for wild beasts the teeth are certainly important but not vital; they can at need secure nourishment without them." . . . "These organisms show a relatively weak cement in the teeth, especially is this true of beasts of prey."

This seems to Adloff entirely senseless; to contrast teeth of herbivora as reaction-capable with carnivora with less reaction-capacity is entirely impossible, since an equivalent stimulus is not in question. The difference in the cement deposit in the two wholly different animal types is to be explained by the two wholly different functional and mechanical stimuli. In the herbivores, we have the greatest mobility in the temporo-mandibular joint, which in higher mammals is differently placed and thus conditions a wholly different construction of the joint; while the carnivore has a simple hingejoint, which naturally must work quite differently. Even the crown cement covering in herbivora is not for the purpose of drawing the crown into the retention apparatus; but the motive is plainly to form from the three substances, cement, enamel and dentin a grinding surface on which the silicic acid, containing, hard grasses may be especially well ground.

So naturally, it is also wrong when Kronfeld says that a herbivore without teeth must starve while man and the wild beast can get their food without teeth. Man, yes; for he can order a prosthesis prepared; but a wild beast without teeth must perish even sooner than the herbivorous animal. The latter finds his sustenance everywhere; he licks it up with his lips, and the hard roof of the mouth is provided with palatine ridges. The wild beast must hunt its food, must kill, for which the canines are needed, must tear, and finally break and crush the bones. But this is all in theory. In reality, every wild beast without teeth is doomed to destruction.

Kronfeld's utterances are not well-grounded. The question whether resistant or not should mean that the teeth under the same conditions of quantitative and qualitative stimuli act at one time in a certain way, at another time, quite otherwise. In comparison of different forms of life the question loses all significance.

Transl. from article—H. A. R.

26—Bauer, Dr. Wilhelm and Dr. F. J. Lang. **"Bundle-bone."** Vrtljhrsschr.

f. Z., 1928. Abs. Brit. D. Jour. L: 152-153, Feb., 1, 1929.

Drs. Bauer and Lang take up the question of so-called bundle-bone raised by Drs. Stein and Weinmann and come to the conclusion that there is no such phenomenon as a physiological movement of the teeth in a horizontal direction, nor any continuous eruption of the teeth throughout life as described by Gottlieb. Horizontal movement of the teeth can only take place when a space is created in the surrounding tissues. This is the work of osteoclasts which widen the space, whether as the result of inflammation (pathological movement) or the removal of lateral support (contact points and neighboring teeth) in which case the physiological stability of the jaw is disturbed. Horizontal movement will continue as long as the inflammatory momentum exists, or where any disturbance such as extraction has caused a transformation in the inner structure of the jawbone and where functional factors (articulation) favor this and render the conditions unstable. Vertical movement takes place in teeth without antagonists and is the result of a stratified deposition of bone on the floor of the socket due to the relief from pressure. The bundles of fibers of the lateral ligament are stretched bringing about deposition of bone on the sides of the socket of the tooth. The moving tooth is always passive. A physiological movement in the sense of Stein and Weinmann does not exist. The deposited bone in the direction of tension in moving teeth in alveolar bone involves with it the fibres of the ligament and corresponds entirely with the bone described by Ebner as "reticulated bone" and is unnecessarily characterized as "bundle-bone" by Stein and Weinmann, who were also misled into stating that this kind of bone was not to be found associated with unerupted teeth or dermoid cysts, or teeth that had not reached the plane of occlusion. Drs. Bauer and Lang have found these layers of bone in all these positions. —*Copy.*

27—Beckwith, T. D. and Adrienne Williams. **Regeneration of peridental membrane in the cat.** Proc. Soc. Exper. Biol. and Med., May, 1928. Abs. Cosmos lxx: 1035. Oct., 1928.

These authors have previously reported on regeneration of this structure in the rabbit and guinea (Cosmos lxix:755, 1927) The present work on the cat might be expected a priori to yield results more directly transferable to man. Injury was caused by passing a dental bur through the alveolar process across the membrane and into the tooth. The cats were 4 and 5 mos. old. After the operation the cats were killed with chloroform: 10 days, and 7, 8, 13, seventeen and twenty weeks. Technique followed is already described in Cosmos for July, 1927. In certain of the animals killed after 7 or 8 weeks, it was evident that infection had developed. This had resulted in destruction of tissue adjacent to the site of the cut, and both soft and hard tissues had been involved.

Destruction in turn had been followed by repair. New cementum had been laid down, upon the external surface of the eroded dentin. As this cementum was formed, fibroblastic migration took place upon the cementum surface. Fibroblasts in turn developed into true fibrous structure. These fibers extended across the field to newly forming alveolar bone, and as the bone repaired these fibres became incorporated within it. Repair of the alveolar bone progressed in the usual manner by osteoblastic activity. It is of interest to note that the alveolar tip extended into the area of destruction rapidly, but on the other hand, growth of the bone toward the tooth was a slower process. This resulted in a longer exposed span for the newly formed periodontal fibers. It was evident, however, that the new fibers were becoming incorporated within this bone.

In brief, replacement of true periodontal membrane and not merely scar formation has been shown in the cat following artificial destruction of the membrane.

—*Copy.*

28—Blumentritt. **Die Tiefenverhältnisse der physiologisch Zahnfleischtasche und der physiologische Zahndurchbruch.** Ztschr. d. Z. xxvi: 819-835. 1928.

Since so much has been written about alveolar pyorrhea, the pathological deepening of the periodontal pocket has been made to play a chief role. The work of Gottlieb especially and the measurements of Orban and Köhler in Vienna have thrown some light on the matter. The physiologic perio-

dontal pocket originates at the eruption of a tooth through the oral mucosa, whereby the outer enamel epithelium grows together with the epithelium of the perforated mucosa. Here the author quotes Gottlieb's now familiar work: Der Epithelansatz am Zahne. Up to this time it was commonly held that, at the eruption of a tooth; i. e., the passage of the point of the crown through the mucosa and at the union of the outer enamel epithelium with that of the mucosa the physiological pocket was already there, and the floor of the pocket was at the cemento-enamel junction. Emphasis is to be laid on the fact that, normally, the pocket floor reaches the cemento-enamel junction much later than heretofore supposed. This stage is reached in most of the teeth of a normal dentition at 30 yrs. From the tables of Orban and Köhler, at 40 years it is not yet reached by all the teeth. The pocket floor is never found at the cemento-enamel junction around the entire circumference of the tooth.

—*H. A. R.*

29—Bodecker, Charles F., DDS. Concerning organs affecting the eruption of the teeth. Int. Jour. Ortho. xiv (8) 657-666. 1928.

Little is known of the forces controlling eruption of the teeth and less of its causes. Sometimes teeth are retained even when there is sufficient space. *Robinsohn of Vienna offers a theory which seems to explain some of the problems.

Before analyzing this theory, the author considers the embryology of the teeth and the surrounding tissues. He describes (1) the ectodermic formations leading to the development of the enamel: these are, the dental ridge, the dental groove, the dental lamina, and the enamel organ. The formation of the dental papilla follows with the beginning of calcification.

To return to the theory of Robinsohn: the existence of the epithelial structures in the peridental membrane have long been known, but little understood. Noyes believes them concerned in formation of cementum; others think them concerned in shaping the crowns and roots, and pathologists consider them responsible for the formation of cysts. Robinsohn develops the idea that these structures influence the movement of the teeth through the bone by the secretion of a hormone under the control of the endocrine glands.

Prof. Bodecker then discusses the two most common theories concerning the factors involved in tooth eruption. Gottlieb, Sicher, and others believe in two such factors: (1) root growth, and (2) pressure of the dental papilla. Robinsohn, however, believes that the chief factor in forcing the tooth outward is the growth of bone surrounding the apex, in conjunction with a resorptive process about the crown.

The author then describes the progressive formation and eruption of a tooth and shows the distribution of the epithelium, etc., by means of diagrams adapted from Robinsohn. Finally he states Robinsohn's theory in full with a critical discussion: R. claims (1) that the epithelial structures of the peridental membrane are in connection with the endocrine glands; (2) that they secrete a hormone which induces a resorption of the bone; (3) that these structures are divided into two parts: crown and root portion; (4) that quantitatively the structures in the crown are greater than in the root; (5) that as a result of No. 4, the bone resorption qualities of the crown areas are more active than those in the root areas.

In conclusion Dr. Bodecker suggests, that if it be true that retardation of eruption may be caused by functional disturbances of the epithelial structures together with endocrine activity, then possible means of regulating such a condition will be found. In the senile extrusion of the teeth also, if Robinsohn's theory be correct, these structures may be re-stimulated to normal function.

Means for accomplishing this are, however, not yet at hand; light therapy is said to promise results.

* *Robinsohn, Isak: Ztschr. f. Stomatol. xxiv (1-3) 1926.*

—*H. A. R.*

30—Boegler, Edith, D. M. D. Cyst formation on granulation tissue of chronic periodontitis. Dtsche. Mschr. f. Zahnheilk. xlvi: 574-579. June, 1928.

Opinion is divided as to the origin of cysts. Römer and Witzel place the start of cyst formation in a focus of the broad epithelial layers. According to Partsch they originate through fatty degeneration of connective tissue and fatty disintegration of

granulation tissue. Euler thinks both possible but that usually both are active factors.

The author presents 2 cyst formations which seem to offer a further possibility. The first case is a double cyst from a left lower second molar with a thickened connective tissue capsule. In the connective tissue at the apex are strong epithelial cords and tufts; and in one place narrow clefts occur between the epithelial cells. Somewhat further from the apex, separated from the first cyst by masses of connective tissue lies a second cyst which presents the typical picture of a "ripe" cyst; the connective tissue capsule, the investment with several layers of flattened epithelium and the usual cyst content, i. e., disintegrated tissue masses and crystals of cholesterin. In another section the divided connective tissue is seen having penetrated between the two cysts by epithelial cells and granulations. This has the appearance of a growth center whose proliferations tend toward the cyst.

The second specimen consists of a tissue proliferation about the root apex of a left upper second premolar. The tooth whose crown was entirely destroyed by caries was extracted without special clinical findings. The apical proliferation remained localized at the apex and showed macroscopically a horny formation. Microscopically was found a cyst containing epithelium seated close to the apex. Further off is an epithelium free necrotic focus which appears to be a broken-down cyst. From this, separated by connective tissue, lies an encapsulated necrotic focus containing granulation tissue. Cholesterin recognizable in the spear shaped spaces. No epithelium.

Römer calls this type "cystis spuria" and considers it incapable of ever developing into a cyst.

Author's conclusions are that it is possible that epithelial processes from the epithelial containing cysts grow out and may surround and encapsulate the necrotic foci; and that in this manner we may have formed a cyst invested with epithelium. This manner of development is apparently less usual than that described by Römer in which the original spaces arise by disintegration of the epithelial cells in center of the broad epithelial layers.

—*H. A. R.*

31—Box, Harold Keith, DDS., Ph. D., The cementum cuticle. Jour. Dent. Res. viii: 427, June, 1928.

The cementum cuticle is a thin, homogeneous, keratinized layer formed by the crevicular epithelium during its proliferation along the cementum to cover the detached connective tissues. It holds for a time the cementum and the soft tissues in a state of organic union. With the separation of the epithelium from the root, this translucent pellicle remains, as a rule, firmly adherent to the tooth. It is continuous with that important component of Nasmyth's membrane, the secondary enamel cuticle of Gottlieb, and is its histologic counterpart. As it is formed in the same manner on the surface of the root as on the surface of the crown, Gottlieb has termed this layer in its entirety, "cuticula dentis." Weski confirms the existence of this dental cuticle and has termed it "epithelial cuticula," and further he divided it into a crown-part, "enamel cuticula," and a root-part, "cementum cuticula." This division, from a topographic standpoint makes for clarity in periodontal nomenclature and teaching. It would appear, therefore, that this distinction, in addition to the fact that the term "cuticula dentis" is commonly though incorrectly used as a synonym for Nasmyth's membrane, presents obvious advantages in the anatomic division of the dental cuticle. Through its peculiar physical and chemical characteristics, the cementum cuticle becomes at once a most important protective medium for the cementum. Its close adaptation to the root-surface, its toughness, and its special resistance to strong acid and alkaline solutions, endow it with distinctive defensive qualities; and unquestionably the cementum is greatly protected thereby from the fluids and bacteria of the pocket.

—*Copy.*

32—Box, Harold K. Microscopic studies of periodontal pocket.: Proc. Amer. Assn. Dent. Schs. 5th Annual Mtg. 1928, pp. 134-136.

Dr. Box found in his study of the mouths of sheep that fully 90% of them develop periodontal diseases. Dr. Snell suggests it as a matter for consideration as to why the remaining small percentage of sheep do not develop periodontal lesions. Dr. Box presented not long ago some aspects of the relationship of periodontal disease as a focus

of infection; and shows cuts demonstrating the development of what are known as capillary beds opposite the points of irritation by calculus and states that this establishes the fact of easy access to the blood stream owing to the multiplication of capillary loops.

Dr. Box has found that on the cementum there is developed a horny layer of epithelium. "That is, the epithelium attaches itself to the cementum, and this layer of epithelium which becomes horny, he speaks of as the cementum cuticle. Dr. Snell regards this as helping to establish faith in conservative treatment of periodontal lesions, acting as a protection, and preventing "pus-soaking of the cementum."

—*H. A. R.*

33—Box, Harold K. **Dental and periodontal foci of infection.** Oral Health xviii (3) 141-148. 1928.

There are three possibilities of the genesis of focal infection in connection with the dental system and its adnexa to be considered: (1) with disorganization of pulp comes microbic invasion through the root canal into the apical regions where there is an abundant lymph and blood supply to take up the bacteria; (2) overstressed teeth show alterations at the apices which are signs of degeneration; and at such points the teeth are less capable of resistance against the micro-organisms flooding the circulation; (3) paradental disturbances, which resemble morbid processes taking place in the tonsils, are a source of infection. The author goes on to discuss the pocket formation at the neck of the tooth and its histology, and supports his deductions by pathohistologic photographs. The periodontal pocket in paradentitis offers the most favorable prospect for chronic infection; its thin epithelial layer and abundant vascularization are the most prominent factors. Chronic irritation plays an inportant role, and may give rise to prolonged inflammatory conditions which favor the entrance of bacteria into the deeper tissues. *H. A. R.*

34—Crouch, S. Stewart, D.D.S. **Some morphological changes in the jaw bones produced by orthodontic treatment.** Dominion Dental Jour. xl (12) 375-378. 1928.

This paper is merely a summary at considerable length of a paper presented by Dr. Milo Hellman, New York (no date or reference given) with what the author is pleased to call "additional evidence." In addition to main points of Hellman's summary, the author adds "a few points to be emphasized with profit" chiefly in regard to orthodontic treatment.

—*H. A. R.*

35—Feldman, Grigori(y). **Epitheliale Vegetationen bei chronischer Periodontitis, apikalen Granulomen und Zysten,** in: D. Mschr. f. Z. xlvi (24) 1928. Abs. Ztschr. f. Stomat. xxvii: 491, 1929.

1. The epithelial paradental debris of Malassez, preserving the morphologic and plastic qualities of epithelium, under the influence of inflammatory stimuli begins to proliferate and may become the source of epithelial cysts and granulomata.

2. The epithelial vegetations in dental granulomes and cysts may likewise arise from the epithelium of the mucous membrane and this by means of fistulae and periodontal pockets; or likewise from the epithelium of the antrum.

3. The native soils of the epithelial vegetations in the periapical granulomes and cysts are, in most cases and to all appearances, the epithelial rests of the enamel organ.

Transl. from Ger. Abstract—*H. A. R.*

36—Gottlieb, Bernhard. **Paradental pyorrhea and alveolar atrophy.** Fortschritte d. Zahnheilkunde (5) 398, 1928. Abs. JADA November, 1928, p. 2196.

Since the early description of the diagnostic symptoms of diffuse atrophy of the alveolar process, other noteworthy observations have been made. Within the past year, it has been observed (Weinmann) that a high percentage of patients affected with acute diffuse atrophy showed an approximately 20 per cent lowered specific dynamic effect on protein metabolism. In a study of experiments made on thirty dogs in which marked traumatic occlusion had been produced, many significant histologic changes were produced. These changes showed that epithelial proliferation along the cementum is not influenced by the Sharpey fibers but rather by the quality of the tooth surfaces. Neither is the development of cement hypo-

plasia explained upon the basis of the mechanical-functional theory of Haupl and Lang. The manner in which cementum, dentin and bone are resorbed as well as repaired under various forms of excessive stresses strongly contradicts the advocates of the traumatic occlusion theory. The greater number of the cases of alveolar pyorrhea are of filth origin. They are treated by scaling and thorough mouth hygiene. The balance of the cases, in which tartar and filth are absent offer the real difficulty. This type of pyorrhea is usually associated with diffuse atrophy.

—*Copy of part of Abs.*

37—Gottlieb, Bernard. Brief abs. from Gottlieb's annual review. Fortschr. d. Z. May, 1928.

One problem hitherto unsolved is the cause of the deep epithelial proliferation. While Gottlieb thinks that the quality of the tooth surface determines this, since the degree of calcification plays an important role, Haüpl and Lang, in answer to this question believe that the presence in the tooth surface of penetrating bundles of connective tissue fibres are the cause. It is these bundles of connective tissue fibers which prevent the epithelial attachment (Ansatz) from proliferating into the deeper portions; and the firm connection of the epithelial Ansatz to the tooth surface results likewise by means of the remains of these fibrous bundles.

The finest staining methods have failed to show the least remains of such connective tissue fibre bundles; so that this last claim seems unwarranted. We know that there is an organic connection between epithelium and enamel, where connective tissue does not enter the question. Accordingly the claim that the epithelium can directly unite with the cement surface offers not the slightest difficulty. Hence, the opinions of Haüpl and Lang seem as untenable as superfluous.

Gottlieb supports his own theory by the history of experiments within the last three years on more than 30 dogs. One of these he gives in detail: a young dog immediately after the second dentition had metal caps soldered upon the first right molar in both jaws, which articulated with each other at a sharp angle, and very markedly opened the bite. This placed a stress upon the teeth. In the course of the following 11 months the other parts of the jaw came nearer to closing; so that in the last months, the other teeth were in occlusion. The findings on the Epithelialansatz showed a resorption on the pressure side which reached the cemento-enamel boundary, extended deeply into the dentine, in many sections undermining the dentine. This resorption was repaired by bone, the reparative resorption reached to the amelo-dentinal boundary; there was no epithelial proliferation. On the opposite side there was in both directions an epithelial proliferation along the cementum. During the resorption which must have taken an appreciable time, no connective tissue bundles stood in the way of the epithelium which none the less persisted in its original condition.

This finding is well calculated to serve as a proof that it is not the Sharpey's fibres which hinder epithelial proliferation. This brings us back to Gottlieb's own hypothesis that it is the quality of the tooth surfaces which governs the epithelial proliferation.

Author cites a further epithelial finding in another dog (the experiment lasting 4 months). On the pressure side of the lower molars at one point the resorption has extended clear to the amelo-cemental junction; this process has torn away small portions of enamel from the dentine and arrived at breaking down of the bone. The epithelial attachment at its deepest point has taken a spherical form, shoved somewhat forward against the loosened connective tissue yet without epithelial proliferation. At all other points where the resorption in this tooth has taken place somewhat removed from the cemento-enamel boundary, as it has on the tension side of this tooth, as on the malocluded antagonist, as on the two control molars of the opposite side, everywhere epithelial proliferation has occurred. At the beginning of the experiment there was no proliferation; otherwise no resorption could have taken place.

Haüpl and Lang take the ground that developing cement hyperplasias compel the proliferation, and show a normal narrow cement hyperclasia over a dentinal cusp. Orban calls attention to the fact that such a dentinal cusp should not be described as a cement cusp; thus the entire contention falls to the ground.

Gottlieb contends that epithelium *can*

proliferate over cement hyperplasias. "The largest Zementhyperplasien can naturally die away on the surface, lose their connection with the connective tissue and provoke the epithelium to deep proliferation."

Gottlieb says that the massive hyperplasia of the cementum causes a tightening of the tooth. It is of special interest to know whether the space required for the cement hyperplasia is made by itself through pressure exerted on the surrounding bone.

Haupl and Lang who always prefer the mechanico-functional explanation of processes, thus explain the hyperplasia by the fact that the tilting axis of the tooth is found near the alveolar border, that the root apex makes the widest excursion and that the deposition occurs in the direction opposite to that of the tooth root.

Schwarz has shown how impossible this is, and has proved that the tilting axis of functioning teeth lies at the apex, and is to be found in the apical half of the clinical root.

Kronfeld, however, proves that the increase of the width of the cement against the root tip is to be found even in impacted teeth; that even before eruption already there is increase of cemental thickness near the apex although this portion is ontogenetically younger than the parts nearer the amelocemental junction. He could also prove that cement hyperplasias are already found in the molar bifurcations even before eruption.

Kellner has examined jaws histologically in which the upper molars on one side impinged upon the edentulous alveolar process, while on the other side the molars had antagonists and were consequently functioning. Measurements showed that the teeth that were not functioning had a relatively greater cemental breadth and lesser periodontal space. This shows clearly that the cement deposition cannot be a product of the destruction of the tooth surface.

—*H. A. R.*

39—Hatton, Edward H. and Wm. G. Skillen. **Epithelial changes in so-called pyorrhea.** Jnl. D. Res. viii: 211, Apr., 1928. Abs. No. 3.

In the histological study of material taken from persons suffering from pyorrhea our attention in the past has been concentrated on the peridental membrane, cementum and alveolar process. We, Gottlieb, and others have noticed very noteworthy, changes in the epithelium. At this time we are presenting a number of specimens from which we draw the following conclusions.

(a) The pocket is quite generally lined by deformed epithelium that is fairly continuous; and, as a rule, the connective tissues are not actually denuded.

(b) The epithelium extends along the root for a varying distance toward the apex and is distinctly attached to the cementum beyond the bottom of the clinical pocket.

(c) As a rule, the recession of the alveolar process bears a distinct relationship to the progress of the epithelium; as the latter advances, the bone recedes in proportion.

(d) The degenerating epithelial cells attached to the cementum and in the pocket seem to furnish a matrix into which lime salts are precipitated and therefore are a factor in the origin of calculus.

—*Copy.*

40—Kellner, Dr. Ernst. **Histologische Befunde an antagonistenlosen Zahnen.** Ztschr. f. Stomat. Abs. Brit. Dent. J. xlix: 428. Apr., 1928.

This research was carried out on a patient who had lost the first and second right lower molars, in consequence of which the upper corresponding molars were standing far down towards the lower gingiva and had obviously been out of use for some time. The findings were that the bone bounding the periodontal space is independent of function; that function influences only the cancellous bone; the suspensory ligament is not entirely dependent on function; that the cementum increases in breadth as a result of lack of function, also the periodontal space is smaller than with teeth in function. Where there are teeth on both sides of the periodontal space the processes of absorption and repair go on symmetrically on corresponding sides, in contrast to those spaces where there is tooth on one side and bone on the other.

(Author's own summary.)

The condition of the bony lamellae limiting the periodontal space is independent of the function:

2. The function influences only the retro-alveolar spongiosa.

3. The suspensory apparatus seems to be not entirely bound to the function.

4. With teeth out of function, the breadth of the cementum is larger and this one of the periodontal space is smaller than with teeth in function.

5. Where a periodontal space is limited on both sides by dental substance, resorption and repair are as a rule proceeding symmetrically on the corresponding sides contrasting with cases where tooth and bone build the periodontal space.

—*Copy.*

41—Klein, Dr. Alexander. **Systematische Untersuchungen über die Periodontalbreite.** Zeitschr. f. Stomat. xxvi: 417-439, Apr., 1928.

Summary. In 150 teeth 950 measures had been taken. The measures have been made with the micrometer-scale of the micrometer-ocular No. 2 by Reichert. The figures thus found were converted into millimeters. The measures were taken without exception on axially made sections at the following places: 1 on the alveolar edge; 2 in the middle between alveolar edge and root point mesially as well as distally; 3 near the root point, 4 between root point and bottom of the alveola, 5 at the bifurcation. Though with slight deviations from the central cut, only such places were measured as surely went through the middle of the tooth. Summarizing we may say:

1. With older individuals the periodont is on an average with ½ of its value wider than with younger individuals. The entire average value of the periodontal breadth is 0.23 millimeter with individuals between 20 and 25 years, with older individuals between 40-50 years, 0.25 millimeter.

2. With functional teeth which have stood quite by themselves, thus had on neither side a neighboring tooth, we found the average periodontal-space to be widest (0.28 millimeter). With charged teeth that had only one neighboring tooth on the one side, the average periodontal breadth was less. 0.26 millimeter, with charged teeth having a neighboring tooth on either side a periodontal breadth of 0.24 mm. resulted. Teeth without antagonists showed the least periodontal breadth (0.20 mm.)

3. The average periodontal breadth is the same in the upper as well as the lower jaw, it amounts to 0.25 mm.

4. The removal of the root point from the bottom of the alveola amounts on an average to 0.25 mm.; at the bifurcation we find the periodont to be 0.32 mm. wide.

5. The average periodontal breadth at the alveolar edge (measured in 260 cases) amounts to 0.39 mm., the same in the middle between alveolar edge and root point (measured in 274 cases 0.17 mm.; in the territory of the root point (measured in 224 cases) 0.21 mm. Thus the shape in the alveolar (sic) results as follows: it is narrowest in the middle of the tooth, widest at the alveolar entrance, and shows in the territory of the root point a breadth between these two values.

6. With functional teeth the narrowest place in 74% of the cases is below the middle, in 21.3% above the middle, and in 4.6% of the cases in the middle. With not functional teeth, the narrowest spot was in 54.54% of the cases below the middle, in 31.81% above the middle, and in 13.63% in the middle. Thus it follows that the turning point of the tooth is to be found in the middle part of the root near the root-point.

7. With front teeth as well as with molar teeth the periodontal breadth is at the alveolar entrance and in the middle, in the majority of cases, at the mesial side wider than at the distal one. In the territory of the root point, in the majority of cases, mesially smaller than distally. In the molar teeth however, the average periodontal breadth, with the majority of cases, is at the mesial side smaller than at the distal one. Apically, in the majority of cases mesially smaller. These differences however, are not very great.

8. With measures taken of sagittally cut teeth, the average periodontal breadth amounts in the upper jaw labially to 0.27; lingually to 0.24 mm.; in the lower jaw labially to 0.21, lingually to 0.22 mm. These results are also a proof that the periodontal breadth is determined by the function.

9. Measures taken in animal teeth have proved that with growing teeth of the rats there is a material difference to the stated of the average periodontal breath between lingual side and labial one. In rats the average periodontal breadth amounts to 0.2, in dogs to 0.35. With animals (dogs, pig, rabbit, monkey, fox, flying dog, guinea pig and rat) the greatest registered periodontal

breadth, 1.12, was measured with the pig.
—*Copy of English Summary.*
Copied verbatim.

42—Klein, Dr. Alexander. Zeitschrift der Stomatologie. 417, 1928. **Systematic examination regarding the width of the peridontal space.** Reviewed by Dr. Balint Orban (Chicago), JADA. xvi: 190, Jan., 1929.

The results are: The average width of the periodontal membrane is 0.25 mm. At the alveolar margin, the membrane is wider, (average, 0.39 mm.) narrower at the middle of the root (0.17 mm.) and again wider at the apical portion (0.21 mm.) The narrowest point in the periodontal membrane is a little below the middle of the alveolus. In older individuals the periodontal membrane becomes somewhat wider, with an average increase of one-sixth the width of that in younger individuals.

The author concludes that the breadth of the periodontal space is determined by function.

43—Kronfeld and Ullik. **Brechen auch bei wilden Tieren die Zähne continuierlich durch?** in: Ztschr. f. Stom. xxvi (1), 1928, p. 1099.

The teeth of wild animals as well as the human ones are erupting continually during life. The eruption of the crown is continued fluently into the eruption of the root with varying speed. The physiological result of this fact is the detachment of the epithelium from the surface of the tooth and the altering of the position of the bottom of the pocket towards the apex. The four schemes given by Orban and Köhler can be proved as well for hedgehogs and bats as for the normal human tooth. The assertions made by Neuwirth and his conclusions into which Häupl and Lang joined have been proved to be a false hypothesis. They are not based on any real research work, as it is possible to demonstrate their inadequateness by studying the necessary material.
—*Copy of author's English abstr.*

44—Lundquist, G. R. **Regeneration of gum tissue following gingivectomy with special reference to mitosis.** Jour. Dent. Res. viii: Abs. ii, Feb., 1928.

The exact method of the regeneration of epithelium removed from the mucous membranes of the gingivae has been a matter of considerable discussion. By removing human teeth with the attached gum tissue, at various periods following resection of the crest of the gingivae, as is done in the treatment of pyorrhea, it has been possible to study regenerating epithelium.

From this material it is evident that the surgical defect is covered by an epithelial layer several cells deep pushing out from the margins of the incision. Nowhere does the covering consist of a single layer of cells. Mitotic figures are found not only in the basement layer but also in the intermediate layer. The outer layer does not contain cells in mitosis. There is evidence in the relative age of the mitotic figures that there is some movement of the dividing cells toward the periphery. As far as these specimens are concerned cell division occurs only by the mitotic method. —*Copy of abs.*

45—MacMillan, Hugh W., D. D. S., M. D., **Radiographic and histologic evidence of the functional adaptation of the alveolar process.** J. A. D. A. Feb., 1928, xv: 316-321.

The chief application of the roentgen ray in dentistry is in the diagnosis of pathologic processes in the bone adjacent to the teeth. Pathologic processes are judged in comparison with normal appearance. A normal adult alveolar process is found only in patients whose occlusal surfaces show degrees of wear. Functional stimuli are transmitted to the investing bone only by occlusal surface stresses—not by tooth-brushes, gum rubs etc. These occlusal stresses cause an increased density of the alveolar process which causes an increased resistance to the roentgen ray. This is the first diagnostic sign of a normal process. The lamina dura affords an attachment for the fibers of the periodental membrane and, through them, transmits the forces of mastication to the supporting bone beyond. The lamina dura varies in thickness and density with the amount of compressive and lateral stresses exerted in mastication. In the normal mandible the lamina dura may not exist as such on the negative. The "thin even white line" showing on the negative is the pathognomonic sign of disuse atrophy. The average patient does not present worn occlusal surfaces and the bone is much less resistant to the rays. The general appearance of the bone is that of disuse atrophy. The X-Ray negative shows a pecu-

liar blur resembling the infiltration due to disease. From the standpoint of bone mechanics, the so-called "residual areas" are more often evidences of normal bone repair, and contain no more infection than the shaft of a healthy femur. —*M. M. B.*

46—Moen, O. H., D. D. S., M. S. D. Tissue changes in treated teeth of known history. J. A. D. A. November, 1928. (Also Northwestern University Bulletin. June, 1930.)

Data presented is based on examination of 25 root-filled teeth of which complete history was available including Roentgen rays before extraction and histological examination immediately after. However the small amount of material precludes advisability of drawing definite conclusions.

No connection was found between the completeness of canal filling and radiographic appearance of periapical tissues. In no case was apical portion of canal sealed by root filling and in many filled to end, the full calibre of canal was not filled. In many of unfilled areas containing debris or degenerating pulp remnants periapical condition was healthy both radiographically and histologically. Frequently gutta-percha points were found projecting into vital pulp remnants and this pulp tissue retained its vitality, thus showing a high resistance, yet the smaller the amount of tissue left in canal for reconstruction the more favorable the prognosis. The degree of regeneration depends also on condition of pulp at time of removal and method of treatment. Treatment must be definitely antiseptic and medication comply with biological laws, potent enough to inhibit bacterial growth but not sufficiently strong to impair tissue.

In all cases where the X-Ray indicated a normal peridental membrane vital tissue was found in the apical foramen. This tissue appeared in two types of change, either atrophic or hypertrophic, and seemed to be of a stable character, and slight trophic variations were attributable to lessened function, and in the absence of irritation and infection such tissue does not undergo degeneration by putrefaction. Where tissue in the apical foramen was found vital, vital tissue was found also in the accessory canals and in some instances vital tissue was found in accessory canals while the main canals showed no evidences of vitality. Injury to periapical tissue due to over-zeal in removing all pulp tissue is more detrimental to prognosis than leaving some pulp tissue in situ.

Resorption and calcification seem to be caused by varying degrees of irritation the more severe types being osteoclastic, and some cases showed absorption followed by calcification as irritation lessened after root filling. Calcification in the accessory canals seemed proportionately greater than in the main canal. *C. R.*

47—Neustadt Egon, M. W. D. The peridental unit. International Journal of Orthodontia, Oral Surgery & Radiography. xiv: 1-22. Jan. 1928.

The peridental unit consists of alveolar bone, peridental membrane and cementum. Of common derivation in mesoderm, these constitute a functional unit. Their range of adaptibility and response to functional and environmental influence is in marked contrast to the immobility of the ectodermic enamel.

Author first considers three structures separately as to anatomy, histology and physiology and goes at length into response of each to normal functional stimuli; later comparing these normal stimuli to the forces and stresses connected with orthodontic treatment. Character of alveolar bone is the same as that of balance of jaw and changes initiated here are projected throughout the masticatory bases and even include the temporo-mandibular apparatus. Bone is most plastic of tissues and responds to mechanical stimuli with ease and rapidity (in marked contrast to slow absorption of gingiva).' In cementum normally resorption and replacement are continually taking place but with abnormal function, permanent absorption or hypercementosis may take place.

The peridental fibres "introduce the factor of suspension into the peridental unit converting occlusal pressure into peridental tension"; and here again inter-dependence of tissues is noted, the functional stresses on peridental membrane producing increased tension in region of attachment of fibers to alveolar bone and cementum with resulting changes.

Chief biologic change under influence of orthodontic appliances is decrease in function—1st, because patients eat softer foods;

2nd, because the appliance prevents to a degree the physiologic mobility of the teeth. The minute movements of tooth in its alveolus, stretching the fibres which in turn engage the alveolar bone are of vital importance and the more rigidly the teeth are held the greater the interference with this functional mobility and the more severe the disturbance of the unit, with eventual atrophy of bone, absorption of cementum and destruction of peridental membrane.

Another change is due to a new force introduced—that of pressure, a rearrangement of trabeculae resulting and a kind of "pressure bone" abnormal to the jaw. And still another element is change in the direction of force which is chiefly horizontal now instead of approximately vertical.

Ideal movement in orthodontic treatment should be exceedingly slow, with apliance permitting as nearly as possible full function to teeth during treatment. This he likens to the physiological mesial "wandering" of teeth throughout life (Gottlieb). In this connection it is suggested that tissues will probably tolerate forward movement of teeth by mechanical appliances better than posterior movement.

In root absorptions not only local forces but also contributing health factors have to be considered and it was brought out in the discussion that in cases of high general resistance and good nutrition, repairs of even extensive cemental absorptions may take place. A nice comparison is made between the stimulus of the act of mastication, and the force exerted by the appliance. In masticating the pressure at the beginning of the stroke is moderate, gradually increasing in intensity and finally towards the end of the stroke, decreasing in force. With the appliance on the other hand, there is no moment of intense application and subsequent relaxation and also there is not the element of periods of alternating exercise and rest.

When orthodontic forces are intermittent the variation is so great as to disturb action of cells which have already been mobilized. This is all in line with general principles of correct exercising. The influence of orthodontic appliances on the tissues of the peridental unit consists of a number of stimuli and as these stimuli vary from the normal they may take rank anywhere between the physiologic and pathologic.

"The reaction is always commensurate with the stimulation. Structures are created by function and for function. The appliance introduces a new, and abnormal function, but a function nevertheless and the tissues of the peridental unit will respond to any function, normal or abnormal."

Discussion by Drs. M. Dewey, F. R. Blumenthal, Henry Ferris and Albert Ketcham emphasized the importance of correlating scientific findings, and practice—the relation of diet and general health to response of "peridental unit" and the use of extra exercising devices to substitute absent normal functional stimulus.

—*C. R.*

48—Noyes, F. B., B. A., D. D. S., Sc. D. **Lymphatics of the dental region.** J. A. D. A. Oct., 1928. p. 1911.

Dr. Noyes reviews briefly the published work of Schweitzer of Berlin 1909-1911; his own done in collaboration with Kaethe W. Dewey in 1915, and that of E. W. Fish of England a decade later, by which experiments lymphatic vessels were shown to be present first in the pulp then in the peridental membrane, in the dentinal tubuli, and finally Gerota fluid was demonstrated as passing through an exposure in pulp and out through apical foramen into peridental membrane, infra-orbital and inferior dental canals, along facial artery to first lymphatic gland of submaxillary chain.

Noyes then describes in detail and by means of diagram his plan of attacking the problem of dental lymphatic circulation from a new angle. In this experiment he has attempted to force Gerota fluid through the tubules of the dentine of a young dog's tooth into the dentine-covered pulp, thence into the lymphatic vessels.

The dissections which would show the results of experiment had not been completed up to time of writing present article but excellent results are to be expected from such a carefully made experiment.

Scopalamin, morphine. and ether were used to anesthetize the dog. —*C. R.*

49—Orban, Balint, M. D. **Contribution to the knowledge of physiological processes in the periodontium,** in: Ztschr. f. Stomat. Oct., 1928. Abs. Brit. D. J. L. 152, 1929.

Dr. Orban in this essay takes up the question of the spaces filled with a connec-

tive tissue, poor in cells and containing bundles of nerves, blood and lymph vessels which Box has described as "a new tissue." The shape of these spaces changes according to whether they occur on the distal or tension side of the tooth where they are elongated, or on the mesial or side of pressure where they are more rounded. On the distal side of the tooth they lie in a recess of the bone with what Stein and Weinmann call "bundle-bone" lying above and below while behind the space there is only lamellar bone. Dr. Orban considers the reason for this to be that no fiber of the periodontium can penetrate through these spaces, therefore there is no possibility of the formation of the so-called bundle bone. The bays or recesses in the alveolar bone are not the result of absorption but are formed as the result of the presence of blood vessels and nerves preventing the formation of bundle-bone which is formed above and below them. They are, however caused by the movement of the teeth and may be traced from the margin of the alveolus to the apex of the root, with layers of bundle-bone between them, often interrupted and replaced by layers of newly deposited lamellar bone. These spaces can be surrounded by bone and act in such cases as medullary spaces. On the mesial or pressure side the conditions are quite different. These spaces, containing vessels and nerves, unite with the medullary spaces when the latter are opened out in the course of absorption due to pressure from the movement of the teeth but the spaces do not cause absorption although their enlargement is the result of absorption. These physiological processes, according to Dr. Orban must be clearly distinguished from pathological ones.

—*Copy.*

50—Orban, Balint, M. D. and J. Weinmann. **Die ursachlichen Bedingungen für den Abbau der Hartsubstanzen,** in: Virchow's Archiv. cclxvii (2) 446-455. 1928.

Summary:

1. It does not appear that a narrowing of the periodontal space through bone building is followed by a resorption in another portion of this space as a result.

2. The fact that the resorption of portions of decalcified tissue takes place significantly slower than in calcified is plainly to be referred to different qualities of the tissues undergoing resorption, of which decalcification is one quality. —*Copy.*

51—Orban, Balint, M. D. **Epithelial rests in the teeth and their supporting structures,** in: Proc. Fifth Annual Meeting, Amer. Assn. Dent. Schools. Mar. 26-28, 1928.

The author shows by beautiful microphotographs the possible relations of epithelial rests in the pericementum and pulp to dentin and cementum formation. The epithelial rests he thinks come from the primitive enamel organ and are found continuously at the apex of the forming tooth. He believes with Bruenn, that these cells govern the formation of the pulp and dentin and possibly the cementum. The presence of these cells in alveolar bone away from the pericementum indicate a shifting of the tooth. The presence of these cells in the pulp may explain denticle formation and possibly the formations of cementum in the pulp chamber. The presence of these cells in the pericementum may explain hypercementosis and cementicle formation in the pericementum. Some epithelial cells to be found in the alveolar bone away from the roots probably come from the dental lamina and represent possibly unformed supernumerary teeth. —*H. J. L.*

52—Orban, Balint, M. D. **Tissue changes in traumatic occlusion,** in: J. A. D. A. xv. 1928. Page 2090.

This article represents a report of microscopic studies made of teeth obviously under excessive occlusal stress. As a result, the author comes to the conclusion "that the whole question of traumatic occlusion is not local, but a question of the physical condition of the individual." He refers approvingly to Gottlieb's observations based on histologic study, that the resorption of alveolar bone and destruction of the pericementum in traumatic occlusion depend on the physical condition of the patient. In some individuals, normal occlusion will traumatize; in others it will lead to the strengthening of the teeth through the development of cementum hypoplasia. He expresses the belief that it is not possible for teeth that have been in good occlusion for years to suddenly develop traumatic occlusion. Where it does occur it appears as a secondary factor. The occlusion should be cor-

rected by grinding down the teeth. Lost teeth should also be replaced in order to preserve those remaining.

The article is profusely illustrated and well worth careful study, as it represents original research into a subject about which there has been much speculation.

—*A. H. M.*

53—Orban, Balint, M. D. **Growth and movement of the tooth germs and teeth,** in: J. A. D. A.—xv (6) 1004-1016. June, 1928. Rev. Brit. D. J. xlix: 864-5. Aug. 1928.

Orban controverts the view that there is any inward or down growth of the epithelium into the connective tissues in the earliest stages of development or during the formation of the root, but holds that the epithelium remains in its original place while the surrounding tissues grow. The roots develop towards the occlusal plane, the epithelial sheath of Hertwig being a fixed point. He also gives a new view of the rise of epithelial rests from the sheath of Hertwig. —*Copy.*

54—Orban, Balint, M. D. **Physiological movement of the teeth; considered from the histological viewpoint,** in: J. Dent. Res. Vol. viii: 7-8, February, 1928. Abs. Proc. Intnt'n'l Assoc. for Dent. Res.

The histological evidences of tooth movement, in the bone of the alveolar process are noteworthy. These observations deal only with the permanent teeth after they have come into occlusion, and are concerned with two types of movement. One, an occlusal movement of the teeth is so marked as to incline one to the belief that tooth eruption is a continuous process. Three stages of this movement may be noted; the first with only partial uncovering of the enamel cap; the second with the enamel cap entirely exposed; and the third, in which the clinical crown includes not only the whole anatomical crown, but also part of the root. Further evidence of this type of movement is seen in the distribution of bundle-bone in the apical region, and in the presence of epithelial cords deep in the bone canal of this same region. In addition there is a gradual movement of the teeth mesially, to account for wear of the contact points. On the mesial aspects of the teeth, the bone of the cribriform plate of the socket is uniformly undergoing typcial resorption; whereas, on the distal aspect, new bone in the form of bundle-bone is being produced to compensate for the loss of bone on the opposite side.

Frequent reference was made to the work of Gottlieb of Vienna. —*Copy.*

55—Schneeberger. **Beitrag zur Pathologie des Interdentalraumes,** in: Schwz. Mschr. Mar. 1928; Brit. D. J. xlix: 481, May, 1928.

Author describes the normal interdental space; then its acute and chronic affections.

Acute: Malocclusion, post-extraction, traumatic occlusion.

Chronic: Chemical injury (acid solutions), calculus, improper hygiene, imperfect restorations or reparative work, etc.

In proof of the foregoing author has experimented on animals with overhanging cement or amalgam fillings with the following results.

Such fillings lead to "niches" in the interdental spaces and thus to unhygienic conditions. Retained food particles and foreign bodies by further irritation of the gingiva cause a chronic inflammation of the papilla. There is no regeneration of the affected tissue. Bone involvement follows with osteolysis and osteoclasis. The interdental bony septum, and with this the most important support of the tooth, is lost. The result is paradental disease. In the dog there is a relation between the degree of disturbance and the kind of filling material; cement causes the most disturbance, amalgam less, and the gold ring the least pathologic alteration. Transl. fr. Lewinski, Ger. reviewer.

—*H. A. R.*

56—Schneeberger. **Beitrag zur Pathologie des Interdentalraumes.** Schwz. Mschr. Mar., 1928. Brit. Dent. Jnl. xlix: 481. May 1, 1928.

"Dr. Schneeberger describes the pathological conditions of the interdental space arising from overhanging fillings and shows the stages of degeneration in radiographs, stating that the degree of damage depends upon the material of which the filling is composed. That cement fillings cause the worst effects, amalgam less bad, and gold rings the least bad." —*Copy.*

57—Sicher, Harry. **Histology,** in: Fortschr. d. Z. (4) 255, 1928.

Author reviews the normal histology of the pulp. This is followed by a discussion of the parodontium. With a series of very fine photomicrographs, he shows the direction of the peridental fibers at the different levels of the root. The illustrations show clearly that the peridental fibers become imbedded in new bone deposited on the "pull side" of the moving teeth. This observation was first made by Stein and Weinmann. The author denies the existence of a circular ligament, but affirms the existence of a very strong interdental ligament. This ligament (a very strong group of fibers) is found regularly between neighboring teeth, just above the alveolar crest. It persists even though teeth are separated through wandering. It disappears only after extraction. —*H. A. R.*

58—Stein, J. B., C. F. Hinck and M. M. Hoskins. **The anatomy, physiology and chemistry of Nasmyth's membrane,** in: D. Cosmos, lxx, 592.

The first half of the article is given over to a consideration of the history of Nasmyth's membrane from the first published report of its discovery by Nasmyth in 1839 to the more recent studies by Tomes in 1882. This is followed by a review of the development of the dental tissues and the opinion expressed that the membranous capsule of Nasmyth is of ectodermic origin. Investigations regarding its histologic nature showed "that while the membrane originally consists of a structureless pelicle and a layer of epithelium, the cells of the latter do not persist throughout life on the exposed surfaces of the erupted tooth. They are to be found only on the protected surfaces where they show evidence of degeneration." The explanation of this is to be found in the fact that there is no mechanism within the membrane itself for the replacement of cells that are worn away by the use of the teeth. Chemical analysis of the membrane showed it to be composed of a mixture of proteins, part of which were glycoprotein.

—*A. H. M.*

59—Solowjew, P. and Winogradowa. **Ueber die Rassenfaktor in der sogenannten Alveolarpyorrhoe,** in: Z. Rdschau xxxvii (47) 1955. 1928. Abs. Fortschr. d. Z. v: (2) 438.

In human beings with different blood grouping, diseases run a differing course. Malaria attacks preferably individuals with blood groups A and AB. Tuberculosis prefers those with Group O while progressive paralysis shuns group O. Investigation of 386 persons with alveolar pyorrhea gave the following results:

Blood Groups	*Without Pyorrhea*	*With Pyorrhea*
Group O	33%	30%
Group A	51%	45%
Group B	11%	17%
Group AB	5%	8%

These findings are not sufficiently marked to determine the predilection of pyorrhea alveolaris for a specific blood group. Russians and Jews were in like case.

Transl. abs.—*H. A. R.*

60—Thoma, Karl H. DMD. **Relation of Nasmyth's membrane to periodontoclasia,** in: Jour. D. Res. 1928. Abs. 19, p. 223. Apr. 1928.

The author presented photomicrographs showing conditions at the base of the gingival crevice and the manner in which the crevicular epithelium is attached to the tooth. At the base of the gingival crevice, the epithelium gives attachment to Nasmyth's membrane (enamel cuticle) which completes the protective lining of the gingival crevice. "Periodontoclasia . . . may be prevented to a great extent by preserving the crevicular tissue. It seems not at all unlikely that the use of abrasives on the rubber wheel, brush wheel or orangewood stick by the dentist may tear away or injure the enamel cuticle under the gum margin, breaking the continuity of the protective membrane lining the gingival crevice. While disease may not follow at once, such injury certainly represents a permanent defect, which leaves a path open to infection." The wisdom of scaling tooth surfaces with sharp instruments in normal mouths should also receive thought in the light of the presented facts. —*Copy.*

61—Turner, J. G. **The pathology of pyorrhea,** in: Dent. Sci. J. Austral.—viii (2) 64. 1928.

Author gives important clinical facts in connection with pyorrhea.

1. Not found under natural conditions in the lower animals, but appears in dogs and

monkeys fed in captivity. It is a disease of cilivization due to refined food. Cites report of skulls found in antiquity. Pyorrhea is a filth disease, especially of youth (?). A sound body is a valuable factor in resistance to it.

2. Extraction cures the disease; but in cases of gouty diathesis this measure fails.

3. Author divides disease clinically into "dry" and "suppurative" cases.

4. As his last clinical point the author stresses lack of cleanliness and scores use of fine, pappy food.

He then discusses bacteriologic and histologic findings. —*H. A. R.*

62—Weissenfels. Erkrankungen des Zahnbettes (Paradentoses) (Diseases of the dental socket.), in: Fortschritte der Medizin. xlvi: 438. May, 1928.

The author discusses first dental caries; afterward subject covered by title. The dental socket embraces a number of different tissues, especially mesodermal, i. e., the cement, periosteum, alveolar bone, gingiva. Here occur the processes of disease spreading chiefly from the neck of the tooth to the deeper parts leading to the final expulsion of the tooth. The attention of the profession has been too largely paid to symptoms—hence the name "alveolar pyorrhea." Lately many investigators have busied themselves with problems of pathogenesis; but these are not yet solved.

The chief factors are endogenous; endocrine imbalances, metabolic disturbances; traumatic occlusion; inflammations, infections, etc. All these play a part. The enormous devastation caused by such disease is a spur to the efforts of scientific workers. Author concludes by pointing out that the presence of infected pocket content, and its continued swallowing may do great harm. —*H. A. R.*

63—Welikanowa. Zur Frage der Histogenese der Nasmythschen Membran, in: D. Mschr. f. Z. H. 20, 1087. 1928. Abstr. Ztschr. f. Stom. xxvii: 492-493. 1929.

1. The outer enamel epithelium (Gottlieb, Orban, Köhler, Gross and Adrion) does not play the predominant role in building the enamel membrane of the tooth. According to investigations of Walkhoff, Adloff, Muenchs, and Reichenbach, as well as the author's own researches, these cells disappear even before the eruption of the teeth and the building up of the enamel membrane.

2. The "ameloblast cuticula" of Koelliker, Ebner, Gottlieb, Neuwirth and others does not, in the author's opinion, exist.

The fine, intensively staining and in thick sections continuous line in the region of the inner end of the ameloblast, which is looked upon erroneously by the author as identical with the ameloblastic cuticula, represents in fact the ektoplasm of the cellular stratum intermedium, which surrounds the ameloblasts and changes them into the ground substance of the enamel. (cement substance.)

3. The enamel—or Nasmyth's membrane, which appears as a product of the protoplasm, or rather of the ektoplasm of the stratum intermedium of the cells, is homogeneous.

The cellular structure demonstrated by certain authors (Waldeyer, Neuwirth) apparently is dependent upon conditions of technique.

4. The incidence of tooth eruption has no existent meaning for the upbuilding of the enamel cuticle. The membrane of Nasmyth is already visible in the stages which directly precede eruption. Fully developed teeth also those not yet fully erupted show on their enamel surfaces the membrane of Nasmyth. It is in no way distinct in its structure and origin from the membrane of already erupted teeth.

5. The enamel cuticula takes part in the building of the pathologic gingival pocket since it forms its inner wall. A physiologic gingival pocket does not exist, as the study of development and eruptive processes show.

Transl. from Zilz, Ztschr. f. Stomat.—*H. A. R.*

1929

64—Bauer, W. Ueber Zementikel und Zementikelähnliche Einlagerungen in der Wurzelhaut, in: Vierteljhrschr. für Zhnhkde—xlv: Heft 3, 345-371. 1929.

Author describes masses of cement substance lying free in periosteal tissue; these masses originate in most cases from calcified epithelia which through breaking down of the "epithelial rests" of Malassez have become free.

These bodies may also give rise to calcified thrombi in the ends of the periodontal veins, in which the metabolism is faulty or non-existent. Parts of the cement cuticle or of alveolar bone, functionally, or by mechanical trauma torn off and lodged in the periodontal tissue may become "Zementikel" in the widest sense. Traumatic hemorrhages into the periodontium may also sometimes become calcified and form the nuclei of these bodies..

—*H. A. R.*

65—Hermann, Becks, M. D., D. D. S. **Normal and pathological pocket formation,** in: Journal of A. D. A., Dec. 1929.

Author deplores the fact that so little has been known or utilized of exhaustive work of foreign investigators in the field of periodontology.

Becks reinvestigates the relationship of epithelium to tooth to determine what is a normal pocket. Sixty-two human jaws were studied besides other animal material and he concludes that "normal" pocket depth is one millimeter or less. Becks disagrees with Gottlieb and Orban as to location of gingival crevice or "normal pocket" as he prefers to call it and is more nearly in agreement with work of Weski in establishing the gingival crevice as an intra-epithelial split, and not a separation between the cuticula dentis and the superficial hornified enamel epithelium. He locates the pocket as bounded by the mouth epithelium and the enamel epithelium, more or less degenerated. As degeneration of enamel epithelium proceeds, mouth epithelium proliferates downward to cover the defect and thus the pocket progresses apically; but when atrophy of the free gum margin proceeds concurrently and in correct ratio with the above changes, depth of pocket remains "normal." When this correlation is disturbed from any cause (inflammatory usually), pathologic activity ensues with a deepened (pathologic) pocket. A pocket is considered pathological when normal depth of above one millimeter has been exceeded and this has been accompanied clinically by bone disturbance and inflamation.

Beck disagrees with Gottlieb and his school in their claim that the bottom of the pocket normally remains at cemento-enamel junction no longer than elsewhere, but confirms the finding of the Vienna scientist that proliferation of epithelium starts before the base of pocket has reached the cemento-enamel junction, and that at this time there is already a layer of new epithelium along the cementum.

Until this newly formed highly resistant epithelium degenerates the mouth epithelium does not proliferate apically, and the pocket remains stationary and "normal." Inflammation or injury may initiate a pathologic activity and the base of the pocket move apically.

Becks distinguishes between "paradentosis" and "paradentitis" as regards etiology. The first commences with bone disturbance or epithelial proliferation; the other (paradentitis) starts with inflammation at margin; or this inflammatory process may be superimposed on a primary paradentosis; and paradentitis followed by a pathologic pocket.

In treatment, causes must be eliminated and deepened pocket reduced to normal by some one of the successful methods known at present.

There are fine lines of histological distinction drawn and substantiated by many photomicrographs and article is further elucidated by diagram showing inter-relation existing between "paradentosis" and "paradentitis." —*C. R.*

66—Bercea, Mary N. **The physiology of tooth movement,** in: Dental Cosmos, lxx, 970, 1929.

The author considers the subject under three heads:

(1) Bone growth and bone reproduction. (2) The pericementum. (3) The cementum.

The first takes up in considerable detail the general subject of bone growth by reviewing the theories which have been advanced during the past hundred years. Reference is made to the classical work by Wolff on this subject, in which he developed the theory of functional form of bone and its transformation in normal and pathological cases. It was his belief that the inner architecture of bone is determined by static conditions which follows exact mathematical laws. Investigations carried on by F. B. Noyes on the maxillary bones, confirm Wolff's theory. The one important point on which all investigators are agreed is that

bone is a plastic tissue which is constantly being remodeled.

Regarding the pericementum and its function, Noyes is quoted as stating that it has a threefold function: First, a physical function by maintaining the tooth in its normal relations to the surrounding soft tissues; Second, a vital function, that of forming bone on the alveolar wall and cementum on the root surface; and third, a sensory function, it being the sole organ of sensation for the tooth. In addition to bone building cells, it possesses cementoblasts that are active in building cementum, by which fibers of the pericementum that have been cut off are reattached.

The cementum is described as a connective tissue whose intercellular substance is calcified and arranged in layers around the circumference of the tooth. Its function is to attach to the tooth the connective tissue-fibers which hold it in position and support the surrounding tissues. If for any reason these tissues are detached they can only be reattached by the formation of a new layer of cementum. To accomplish this they must lie in physiological contact with the root surface. The cementum must be regarded as the most important of the dental tissues. It is stated that its vitality depends entirely upon that of the pericementum, implying that any portion not covered by the pericementum is not vital, a statement which the periodontist has repeatedly shown to be incorrect.

The article is made up chiefly of the review and quotations of other writers and is of interest primarily to the orthodontist dealing as it does with the physiology of tooth movement. —*A. H. M.*

67—Delater, Gustave. "A pyorrhée et les tonsilles gingivales." (Reply to Vincent's article with the same title.), in: Revue de Stomatologie, xxxi: 380-382. July, 1929.

Dr. Vincent's theory fascinating as it may appear does not harmonize with either clinical or histologic findings which we have made and reported. It is based upon the existence of an organ and function which we do not believe demonstrated. Can we admit that the gingival interdental bridges have the same value, anatomic and functional, as the lymphoid formations of the digestive mucosa, especially the tonsils? We think not.

Author then give his reasons.

1. The infiltration of the conjunctive meshwork is diffuse, not the follicular variety found in other lymphatic formations.

2. This infiltration for the most part consists not of lymphocytes, but of plasmocytes, with granular excentric nucleus; that is to say, a reaction to prolonged irritation.

3. It coincides with an inflammatory condition of the epithelium. The most superficial strata of flat cells have fallen off, and no longer offer opposition to the ingress of microbes.

The anatomic state of the proximo-dental gingival papillae and interdental bridges shows an inflammatory process, chronic and latent, rather than a histologic formation. This interpretation is confirmed by the presence of a slight serous exudate, about constant in the adult, which may be demonstrated by pressure on the gingiva. Dried, fixed, stained, and examined with microscope, this serosity shows all the characters of pus (which author enumerates).

This is then the state of latent gingivitis.

Would it not seem more reasonable to admit that the gingival folds formed around the necks of real pockets (in miniature) where the debris of food, and microbes accumulate; that these latter find "bed and board," multiply, as a rule neutralized by the leucocytic defense, and creating this latent condition from which they work from without inward.

In order to set up an active process it is only necessary that the defense should be lessened, by traumatism of continued malocclusion, etc., or through a general systematic weakening due to intercurrent disease.

To Delater it seems that the two cases of vaccinotherapy make it clear that the pyorrhetic symptoms were the sequel to an intestinal infection. The microbes of the autovaccines used came from the gingival pus of the patients; only success obtained with microbes of intestinal origin would have been of value, even so it would have remained to prove their route by way of the blood or the "gingival emunctories."

The literature abounds with such cures with vaccines from gingival pus. It is poor in cures long maintained in spite of the persistence of deep peri-cervical cul-de-sacs, foci of microbic retention. —*H. A. R.*

68—Gämän, Dr. F. Beiträge zur Frage der Kieferknochenstruktur unter normalen und einige krankhaften Verhältnissen, in: Ztschr. f. Stomat. xxvii (9) 1844. 1929.

The structure of human bone, says the author, has been thoroughly investigated, and too, we know, not only that the trajectorial structure of bone is characterized by a certain obedience to law and is determined by external mechanical influences acting on the bone; but we also know how the trajectorial system develops under the action of these external influences.

After a lengthy review of the literature and a detailed description of the trajectorial systems, with many interesting illustrations, the author summarizes as follows:

1. Below the teeth under certain circumstances a new osseous framework or stroma may develop in the dental functionally oriented zone.

2. The form and course of development of this trajectorial system is in our experience very much alike in the human subject. From each root apex arises a lightly curved trabecular network whose convexity is strongly directed backward.

3. Its development is always related to various external relations; further the alveoli of the basilar trajectory are well built; in case of teeth used as bridge abutments, in cases of bruxomania, and to a great extent in the remaining teeth of individuals who have suffered losses. This trajectory is not found (or only in slight degree) in teeth whose antagonists have long been absent, in persons suffering from pyorrhea, and in some disturbances of occlusion. Alveoli of the basal trajectory when present, disappear if the corresponding tooth does not fulfill its function, e. g., if we extract its antagonist.

The circumstance that in most cases the alveoli of the basilar trajectory are found in animals in contrast to the human, supports our contention; which brings the development of this trajectorial system in relation with heightened functional demand upon the tooth. —*H. A. R.*

69—Gottlieb, Bernhard. Alveolarpyorrhoe und Alveolaratrophie. Fortschritte der Zahnheilkunde. May, 1929 V (1).

Gottlieb discusses first the work of Box. Under chronic periodontitis of the pocket building type Box distinguishes two forms:

1. Simple. 2. Complicated.

(1) In the former, the sites of election are characterized by about an even distribution in the denture, chiefly due to unfavorable hygienic conditions.

(2) The gingival inflammation rules the whole picture.

(3) The tooth loosening occurs as a late symptom.

(4) Pocket formation is early.

(5) Careful cleansing is often followed by a rapid recession.

2. Complicated form: characterized by:

(1) An unequal distribution of chosen soil, decided by the presence of traumatic occlusion.

(2) Extrusion from the alveolus or sidewise shifting with formation of diastemata an early symptom.

(3) Tooth loosening also occurs early caused by a widening of the periodontal space.

(4) Pocket formation occurs relatively late, localized on the side of tension or pressure.

(5) The gingival inflammation has no great significance.

(6) The teeth are usually in a high degree immune to dental caries; cleansing exercises only slight influence on the degree of loosening. Regulation of the occlusion however has a remarkable effect on tightening the teeth.

Box considers that the pocket building gum does not atrophy whether on different topographical grounds, swelling or hypertrophy; or, that a tooth in traumatic occlusion cannot yield.

A discussion of the treatment of the periodontal pocket follows:

(Periodontoclasia fibrosa and the treatment of the periodontal pocket). Gottlieb then discusses traumatic occlusion which, as he says, plays so important a role in America. Unfortunately the Americans have not yet arrived at facing the problem squarely. A patient presents himself with loosened teeth which are found to be in T. O. The

teeth are ground into occlusion and the condition is improved. Everything points to the fact that T. O. is the important etiological factor. No one asks the question, How does this T. O. come about? If for example, we consider a case of open bite, which in youth presented typical occlusion; when a tooth in such a case is found in T. O., this tooth must have made a pathological change of position. The etiology of the disease is to be sought in the change of place which we call tooth shifting, and recognize as a cardinal symptom of diffuse atrophy. Consolidation of a shifted tooth is only possible when a secondary T. O. is not present.

To return to Box: When, for example, a diffuse atrophy is present at the distal root of a lower molar, and this root is slightly extruded, a tilting movement of the tooth ensues in a mesial direction. If normal contact relations are present this tilting cannot take place; but if the distal part of the molar crown is a little extruded, and the pressure of its antagonist is exerted in a mesial direction, then the tooth will be forced in the direction of a resultant between the force exerted by the adjacent and the antagonist teeth. It is not clear to Gottlieb why this result should work greater harm than when the tooth can yield. Just how it leads to pocket formation is to him incomprehensible.

Box states further that in simple periodontitis the marginal gingival atrophy keeps step with the deep epithelial proliferation; and this fact contributes to keeping the pocket shallow, in which view Gottlieb concurs. The histologic substratum for the clinical picture of complicated periodontitis, is according to Box, the rarefying periclasia fibrosa caused by T. O. There is productive inflammation in the periodontium characterized at first by the presence of plasma cells, lymphocytes and epithelial cells; in time there is a concentric perivascular fibrosis, thickening of the intima and final obliteration. With this view Gottlieb does not agree. His experiments on dogs with Kronfeld have shown that in 24 hours osteoclasts break down the alveolar bone, and in this way, make room for the soft tissue.

Gottlieb describes his own views of T. O.; not an overstepping of a boundary to be expressed in absolute figures. Every denture, every tooth has its own limits which for each may change with time. We may also recognize a T. O. clinically only by the damage already committed. However such teeth often come into T. O. by a primary shifting; now each shifting tooth is loose. Such a tooth was loose already before the traumatic occlusion. Each functionless tooth has a narrower periodontal space than a functioning one. The widening of the periodontal space can only occur through bone resorption.

After detailed study of Box's treatment, Gottlieb considers the question: Does the quality of the hard tissues play a part in their resorption? Bauer and Lang doubt the correctness of the conclusion that in orthodontic measures the cement reacts differently than the bone, and call attention to the work of Ketcham, who studied root resorption in process of tooth regulation. Oppenheim has proven with most desirable clarity that such resorption occurs only on the use of rabid force, and that among many cases regulated by himself and Grünberg without exception no single case could resorption be demonstrated.

Gottlieb continues with a long difference of opinion between himself and Haupl and Lang who try to explain the different processes in bone and cementum by the vascular relations (before quoted); then they say —"The more advanced the decalcification, the more the Sharpey's fibers are decalcified so much more readily does resorption take place. The more vital organic substance the hard tissues still contain, the less likely is resorption to occur."

Gottlieb does not agree with Haupl that the increasing cemental deposition shows resorption going on in the adjacent bone. Orban and Weinmann have shown in rats on experimental diets that cement and bone deposition almost arrive at contact without resorption. Gottlieb shows a photograph in which the periodontal space about a functioning tooth is reduced to almost nil by cement building, without producing any trace of resorption as a result of the narrowest space.

Kronfeld has recently shown that in the milk tooth resorption in various animals (sheep, dog, squirrel) over relatively long intervals, the resorption stops exactly at the limit between calcified and uncalcified tissue; to such an extent does it avoid the latter that entire corners or islands of uncalcified tissue remain unaffected. Stein

found in his *Plantationsstudien* that by implanting the graft in the hollow (long) bones of the host, a proportionate resorption of graft and host bone never resulted; as a rule resorption occurred only in the host bone, although the connective tissue between is homogeneous. Uncalcified grafts showed as a rule only indisputable traces of resorption only where a recalcification of tissue has already begun.

A. M. Schwarz has lately shown that the pivotal center of the functioning tooth lies below the middle of the clinical root.

Gottlieb cites the researches of Alex Klein on the width of the periodontal space. Klien believes that we may speak of a biologic and physiologic space; the biologic is the lesser, as it is found on retained and reaction capable dentures of non-functioning teeth. Through functioning the physiologic breadth is increased.

In the matter of the epithelial attachment Gottlieb goes on to say that the important question is whether the loosening of the epithelium from the tooth surface and its deep proliferation along the cement is to be regarded as physiological or pathologic. He refers to his own earlier studies when his sections showed the deep epithelial proliferation without inflammation, which had however probably ceased at the moment of death and rendered the findings thus insignificant.

Wild animals never show deep epithelial proliferation; hence only on these can the physiologic relations be studied.

Kronfeld and Ullik have carried on these studies on the teeth of animals taken directly from their wild life, and on such animals have found a progressive loosening of the tooth surface epithelium and deep proliferation along the cement, just as in domestic animals and humans with no trace of inflammation. The depths of the pockets were negligible and the horny layer of the mouth epithelium went over without a breach into the horny layer of the tooth.

Haupl has taught that the thickness of the fibrous bundles decreases toward the cement boundary and that, in the deeper parts of the periodontium the density increases and cites Ebner and Wetzel.

Gottlieb scoffs at all this and states that the pocket floor is never found at the amelocemental junction without deep epithelial proliferation along the cement. Haupl holds that it may be proved by many sections that the pocket floor lies either at the cemental juncton or somewhat above it without the epithelial proliferation along the cement.

In regard to continuous cement deposition Gottlieb has stated that the Sharpey's fibers penetrating the cement become calcified in time, lose their functional ability and necessitate a growth of new fibers. A fully calcified fiber is dead and continues functional relations with the periodontium. Haupl regards the entire building of bony substance as opposed to this—"in which the lifeless earthy salts are in organic relation to the cement substance and both in relation to the uncalcified fibrillae." Gottlieb calls this an unfair statement. The fibrillae build a network of organic substance which is not calcified. The meshes of this network are filled with uncalicfied cement substance. Were the fibrillae calcified, and thus the entire bony tissue calcified, then the resistive power of bony tissue would be at an end.

Kronfeld has shown by systematic staining (Blelschowski) that by highly vitalized vegetarian dentures, the uncalcified Sharpey's fibers can be traced deeply into the cement; while in meat-eaters they remain on the tooth surface. In the radical-surgical operation and other operative procedures such as replantation, no fibers insert themselves into the tooth surface; and the epithelium must proliferate into the depths of the denuded root. From this we may perceive that the character of the root surface over which the epithelium must proliferate plays a part not to be overlooked.

After criticism of Schwarz's work Gottlieb goes on to discuss the Dunlop method.

(Abstract of chief points in G's annual review, 1929.) —*H. A. R.*

70—Häupl, Karl. Ueber Gewebsbildung im Paradentium, in: Vierteljahrsschr. f. Z. xlv: 606-618. 1929. Brief abstract in D. M. f. Z. xlviii (19) 1315. Oct., 1930.

The author follows the now familiar consideration of paradental tissue formation with observations on the problem of tooth wandering, the breadth of the periodontal space; and the vascular supply of the supporting tissues of the tooth.

In this article the author sets himself

squarely against the views advocated by Gottlieb on this subject. In the space of an abstract, impossible to do justice to these conflicting opinions.

(From Abstr. in D. M. f. Z. as noted above.) —*H. A. R.*

71—Kelly, J. Albert, B. S., M. D. **Bone atrophy and absorption,** in: Int. Jour. of Orthodontia, Oct., 1929, xv, 949-975.

Bone atrophy is applied to those conditions in which the total amount of earthy matter in a given bone is less than normal and is due to either a decrease in the size of the bone or in the density. Two kinds of atrophy—concentric and eccentric. Bone is constantly undergoing change of substance and architecture throughout life. Bone is a material composed of an organic matrix which is impregnated with inorganic salts, chiefly phosphates and carbonates of calcium. Atrophied bone is lighter in weight, slightly smaller in diameter, and weaker than a corresponding normal bone. Senile atrophy affects practically all of the bones of the skeleton in aged individuals. Cohnheim regards it as a physiologic change while Recklinghausen considers it a nutritional disturbance. It has also been considered to be neurotrophic in origin. Hunger atrophy is due to the deprivation of proper food and is relieved by correcting the calcium deficiency. In scorbutus the bones become atrophic due to the inability of the cells to form intercellular material. There is probably also some disintegration of the organic matrix. This is especially marked around the teeth and causes them to become loosened. This condition is promptly cured by supplying the antiscorbutic vitamine D. Osteomalacia is a condition in which the bones are softened and contain very little mineral matter. There is a negative calcium balance which is not benefited by a high calcium diet or cod liver oil. In rickets the bones are softened. Only growing bones are affected and it can be cured by an adequate diet plus Vitamine D, from cod liver oil or sunlight. Probably many cases of malocclusion are the result of rachitic softening of the jaws. Atrophy of disuse may be expected if a bone is removed from the necessity of resisting mechanical forces. Acute traumatic bone atrophy is of rather frequent occurrence. It involves the bones of an extremity which has been subjected to trauma. The root absorptions occasionally noted after orthodontic procedures are a form of this condition. Atrophy accompanying local inflammation occurs in the vicinity of infections such as joint tuberculosis and is one of the earliest roentgenographic signs of the disease. Pressure atrophy is one of the forms about which there is still much to be learned. It is one of the fundamental principles of orthodontia. Neoplastic resorption is found in bone which is in the line of growth of bone cysts, fibrocystic disease, and benign and malignant tumors. The tumor cells themselves seem to possess the power of dissolving the adjacent bone without phagocytosis or even direct contact. Neurotrophic disturbances of bone occurs in tabes dorsalis and syringomyelia and is believed to be due to the destruction of some of the sensory nerves to the part. Atrophy is the result of excess resorption. Normal bone is the result of a delicate balance between constant resorption and deposition of new bone. (The article is profusely illustrated.) —*M. M. B.*

72—Lartschneider, Dr. Josef. **Die sogennante "Malasseszchen paradentären Epithelnester" und ihre biologische und pathogene Bedeutung. Beiträge zur Kenntnis des Wesens und der Entstehung der multilokulären Kieferkystone, Odontome, Adamantinome und der kongenitalen Epuliden,** in: Zeitschr. d. Stomat., xxvii: 476-480, May, 1929.

The author distinguishes two groups of "paradental epithelial nests" of Malassez (1884), which have nothing in common, either in biologic or pathogenic significance, or in localization. One group lies under the mucosa, between it and the crowns of still unerupted or retained teeth and are the remains of the tooth bands (or ligaments). The other group lies embedded in the periodontium along the roots; hence may be called pararadicular, not paradental. These are groups or clusters of enamel epithelial cells; each represents the epithelial remains of the enamel organ. Such cells may awaken to renewed vitality and give rise to the "enamel pearls."

He divides the ligamentary submucous group also into two groups, but differentiates them none too clearly. These clusters when aroused to activity may help build

more or less fully developed teeth or layers of enamel; or they may give rise to tumors which appear as conglomerates of teeth, enamel, dentin or cement layers. These tumors may change into multilocular cysts, such as are observed in other organs; but there are always histological traces to be found which belong to the enamel or cement layers; so that these are not readily confused with adeno cysts.

The odontogenous tumors of the jaws have nothing to do with tumors in the sense of blastodermic tissue hyperplasias. These do not give rise to metastases, while the odontogenous tumors do.

—*H. A. R.*

73—Lindsay, Mrs. Lillian, L. D. S. **The movement of teeth. A note on certain histological researches of Drs. Stein and Weinmann,** in: (Brit). Dent. Record xlii: 109-110, March, 1929. Abs. Int. J. Ortho. xvi (4) 409, Apr. 1930.

Stein and Weinmann noted that in normal jaws the deposition of a certain kind of bone at the distal surface of the tooth roots was found with unfailing regularity, consisting of bundles of fibers; when it appeared on the mesial aspect of the root, it was in isolated patches. The fibers of the suspensory ligament did not enter the bone on the mesial side, whereas on the distal side they continued into the bone. On the mesial side of the tooth, on the other hand there was found evidence of absorption. Therefore, in the interior of the alveolus, the bone about the tooth roots is built up distally and removed mesially, the result of tension on the distal and pressure on the mesial surface. There were evidences of a labially-directed movement in the maxillary, of a lingually directed movement in the mandible. Where islands of bone were found on the mesial root surface, no movement had taken place for a long time. —*Copy of abstr.*

74—Münch. **Nimmt das aeusere Schmelzepithel an der Bildung des Schmelzoberhäutchens teil?,** in: Vrtljhsschr d. Z.—xlv: 80-83. 1929. Abst. D. M. f. Z.—xlviii (19) 1312. Oct., 1930.

The author has already in previous articles opposed Gottlieb's theory of the genesis of the enamel cuticle, which according to his investigations is no longer tenable. In earlier stages of development vessels enter and penetrate the enamel epithelium. In the author's opinion these vessels have a resorptive feature. His preparations are taken from the dog. Author reserves further discussion of these observations for another article.

—*Transl. copy of abs. H. A. R.*

75—Norberg, Olof. **Untersuchungen über das dento-gingivale Epithelleistensystem im intrauterinen Leben des Menschen.** Vrtlsjhrsschr. f. Z. xlv (3), 498-500. 1929.

In this admirable monograph the author treats of

1. The origin and early development of the primitive tooth germ especially in relation to the so-called lip furrow band.

2. The further development of these structures and their influence upon the architecture of the jaw especially the mandible during fetal life.

3. The developmental structure known as the median and lateral enamel rudiments and enamel niches at a certain period of the development of the enamel organ.

4. The mode of growth of the tooth germ dorsal to the enamel organ of the second milk molar during the formation of the permanent molars, especially the first of them.

5. The so-called substitute enamel organs of the permanent substitute teeth which develop therefrom.

The results (27 in summary) of the author's extended investigations would require too much space but will repay time spent by those interested in consulting the original paper.

The last of these results as summarized by the German reviewer is as follows:

Between the epithelial rudiment on the one hand and the surrounding osseous tissue there appears to exist a certain reciprocal relation in-so-far as, that as the one declines the other develops. This leads the author to think that the dentogingival rudimentary epithelial system, beside its tooth and gum forming function has still another, viz.; to contribute to the fixation and protection of the enamel organ in the mesenchyma until the primitive alveolae and tooth sockets have been developed.

Brief abs. of lengthy Ger. review.

—*H. A. R.*

76—Orban, Balint, M. D., and Emil Mueller, M. S., D. D. S. The gingival crevice, in: J. A. D. A., July, 1929.

The present article is marked by same lucid convincing style which characterizes writings of Gottlieb and others of this Vienna School, and is particularly to be recommended for its logical correlation of clinical practice with histological findings.

Some former writers have considered that the epithelium merely lies close to the surface of the enamel and is attached to the tooth only at the cemento-enamel junction; that the base of the gingival crevice is at the cemento-enamel line. As a matter of fact, up to about thirty years of age the epithelial attachment (and base of crevice) is on the enamel and in later life on cementum, bottom of crevice and epithelial attachment shifting continually rootwards.

At eruption all of enamel except the erupted tip is actually attached to the epithelium, is in fact in organic contact with it. This attachment drifts apically, gradually but continuously throughout life, tooth eruption being a continuous biologic process, though the rate is variable, the slower the process the more favorable to long retention of the tooth.

Epithelium is attached to considerable portion of root surface and this attachment shifts rootwards in advance of bottom of crevice. From examination of hundreds of human teeth it is found that epithelial attachment remains no longer at cemento-enamel junction than any other level.

Gingival crevice is defined as space surrounding the tooth limited on one side by surface of tooth, on the other side by crevice epithelium. Ideal crevice is shown both microscopically and clinically to be very shallow or nil and this shallow condition to be not unusual. Crevices of one to two millimeters or more are not necessarily pathologic, but the deeper the pocket the more likely it is to become pathologic. The crevice develops between enamel cuticle and epithelium, the cuticle being the remnant of the epithelial attachment left after detachment of epithelium from tooth surface.

Much of histological error has come from misinterpretation of photomicrographs, the space originally occupied by enamel but dissolved out in preparation of specimen being mistaken in many cases for the gingival crevice. Microscopic examination clearly shows distinction between crevice epithelium and epithelial attachment. Epithelial attachment is smooth, thin and well hornified and its border cells greatly compressed. Crevice epithelial cells are less compressed, not thoroughly hornified, the membrane is thicker and its contour much less smooth and regular. Box's "breaks in epithelial lining of crevice" described as "weak link" in periodontium can only be artifacts in epithelial attachment. Every point in argument is substantiated by excellent photomicrographs of human specimens.

It is further shown that calculus is deposited on tooth surface to the very bottom of crevice and even healthy gingival crevice cannot be considered "self-cleansing." Black's clinical observations on height of gingivae in children and recession of later life are in absolute conformity with present-day histological knowledge. His misconception with regard to immunity to caries of sub-gingival space was due to the fact that he found that enamel behind the gums in extracted young teeth was free from caries. This surface was not, however, in gingival crevice as he supposed, but still united with epithelium and so protected from mouth environment and contamination. Thus his extension of filling beneath gum margin is based on a wrong idea of histology. In any case "gum margin" is not stationary, and irritation of filling or placing crown beneath margin may hasten its recession.

The gingival crevice to its deepest portion is an unclean place in the mouth; it is filled with calculus, destroyed epithelial cells. leukocytes, bacterial plaques, etc. The shallower the crevice the smaller the amount of accumulations. Healthy gum tissue is not a protection against caries, but healthy gum usually goes along with a shallow crevice. Good mouth hygiene, proper brushing and massage, by helping to maintain or bring about an ideal shallow crevice, constitute best preventive of caries as well as gingivitis and, eventually, suppurative periodontoclasia.

In planning restorations, principles of anatomy, biology, aesthetics and mechanics, all are to be considered, and sometimes extensions are necessary to meet these requirement, but no crown can be extended far enough gingivally to save tooth indefinitely

from decay. Better if possible leave margins where easy to polish and keep clean; and when aesthetics demands extension, realize what is to be expected, and be prepared for readjustment or replacements, especially in young patients.

How can there be recession accompanying inflammation as found in pyorrhea? Infection and inflammation cause shifting of epithelial attachment and bottom of crevice apically, with gum margin at first remaining in position, and thus we have pocket formation. With subsidence of inflammation, atrophy succeeds and gum margin recedes to new low level of epithelial attachment; as is illustrated following successful periodontal treatment.

Recession may be physiologic, due to accelerated rate of shifting of epithelial attachment on root. The slower the rate the more favorable, but the epithelium will always grow along root sooner or later in human beings and other animals with similar teeth, and Gottlieb considers this shifting without pocket formation, physiologic.

The photomicrographs and diagrams accompanying article and the context are well worth careful study. —*C. R.*

77—Orban, Balint, M. D. **Histology of dental pulp and periodontal membrane,** in: J. A. D. A.—xvi (6), 965-996, June, 1929.

This article is a scientific research in histology, with profuse illustrations and extended bibliography. The author's conclusions are as follows:

1. With the proper histologic technic, the different cell types in normal as well as pathologic conditions can be easily recognized and differentiated.

2. In the dental pulp, we find the same cell elements that have been recognized and differentiated by Maximow in the loose connective tissues elsewhere in the body.

3. The same cells are also present in the tissues surrounding the roots of the teeth.

4. Inflammatory reactions occur in the periodontal tissues in the same way as elsewhere in the body. The result of an inflammatory reaction will depend on the exogenous and endogenous factors; the virulence of the bacteria and the response of the reacting mesenchyme. —*Copy.*

78—Orban, Balint, M. D. **Contribution to the histology of the pulp and periodontal membrane with special reference to the cells of defense in these tissues,** in: Jour. Dent. Res. ix (5) 610. Oct., 1929. Abs. No. iv.

Slides were presented showing the presence of histiocytes, undifferentiated mesenchyme cells, and ameboid wandering cells in the normal human pulp and periodontal membrane. On the basis of a specimen of chronic abscess at the apex of a bicuspid, the transformation of these cells into "polyblasts" was shown. The polyblasts of Maximow are phagocytic, mononuclear, wandering cells in the field of inflammation.—*Copy.*

79—Prader, Dr. Florien. **The presence of bundle bone in some anomalies of occlusion,** in: D. M. f. Z. May, 1929. Abs. Brit. D. J. 50: 781, July 1, 1929.

Further illustrations are added to the growing evidence that the tooth socket is a separate entity. Dr. Prader shows that with every alteration of pressure on the tooth or tension, the socket is transformed, that is, absorbed on the pressure side, whilst deposition of new bone takes place on the side of tension. He shows clearly that the two processes of absorption and deposition may take place simultaneously on the same side of the tooth. Four cases are illustrated and discussed; thus, formation of bundle bone, the author suggests, is the result of the inner forces of the alveolar bone. —*Copy.*

80—Reppeto, W. M., D. D. S. **The biologic aspects of periodontal disease as related to orthodontic regulation,** in: I. J. O. O. S. and Rad. June, 1929.

Author attempts "to show the relations of various biologic principles to orthodontic treatment concerning the reactions of tissues to the application of the necessary forces."

It is to be understood that in health various complicated changes and reactions are constantly taking place in the periodontal tissues, developmental first, then functional, and finally those which are the natural accompaniment of later life. Constant adjustments are necessary in the tissues to meet changing demands. Orthodontic treatment of necessity disturbs these biologic and physiologic changes, and the important thing is to conduct treatment without occasioning too much abnormal function and stimula-

tion. The very presence of irregularity induces some biologic disturbance which must be reckoned with in attempting to correct the clinical picture.

Dr. Reppeto goes into the development and histology of enamel. He summarizes, Gottlieb and Orban's conception of the eruption of the teeth, "a continuous biologic process"; and points out that in orthodontic treatment this biologic progression is disturbed, and when treatment is carelessly applied usual biologic processes are modified into pathologic. Thus, the normal process of apical advancement of the epithelial attachment may be so greatly accelerated and with it the proliferation apically of epithelial cells as to be an etiologic factor in periodontoclasia; the attachments of the teeth being materially reduced, their resistance to masticatory stress is lessened, and thus the orthodontic appliance becomes a factor in developing traumatic occlusion. If measures are taken to reduce the various stimuli as soon as objective and subjective symptoms arise adjustments will usually take place in all tissues without much injury and "normal biogenetic forces will again be stimulated to aid in the correction of the abnormality." Without such care correction is likely to be obtained at too great cost, and pathology develop later in life following an apparently good result, the tissues not having been able to adapt themselves to the various types of extra-alveolar leverage brought to bear upon them.

There are different grades of adjusting powers in various individuals which must be considered by the orthodontist, and he must be able to note early in treatment by observing tissue tone the type of reaction he is getting. With such care the effect of orthodontic treatment both locally and generally will be invaluable as a palliative and a prophylactic measure. —*C. R.*

81—Schumacher, Siegmund. Wandering of Teeth of the Chamois and Deer, in: Vrtljhrsschr. f. Z.—No. 4 1929. Review by Kronfeld. JADA. xvii (5), 939-940, 1930.

Stein and Weinmann found that in 1925 in their study of human jaws that all during life teeth migrate steadily but slowly forward. The alveolar bone is resorbed on the mesial side during this process, and new bone is deposited on the distal side. At the same time the teeth are slowly moving towards the occlusal line and as an evidence of this movement we find a deposit of layers of newly built bone on the bottom of the alveolus. Schumacher studied these changes in the teeth of chamois and deer. These animals are herbivorous and their molars are subjected to a great amount of wear (abrasion) on the occlusal surface. It has been observed that in spite of this abrasion the molars of these animals show a fairly uniform length throughout life. As it is fairly easy to determine the age of a deer by the configuration of the horns, the author was able to collect jaws of animals of known age, from early youth to about 12 years of age. He found that each molar is continuously moving towards the occlusal line (vertical wandering).

In youth this movement is compensated by a correponding increase in the length of the root. After the root formation is completed the entire tooth moves toward the occlusal surface. Thus, the distance from the root end to the mandibular canals is constantly increasing. The bifurcation is coming closer and closer to the alveolar margin and in the case of a chamois buck of 12 years was found to have passed the alveolar margin. In a similar way, the horizontal wandering has been studied by Schumacher on the maxillae of bucks of different ages. He found that the entire length of the row of bicuspids and molars is continuously decreasing during life. In young bucks, the length is about 69 mm. and at 8 years it is around 60 mm. This shortening, which is due to interproximal wear, is caused by a shifting of all teeth forward toward the median line. This movement corresponds to the shortening of the human tooth arch during life by the loss of tooth substance at the contact points which is histologically determined by a steady slow movement of all teeth toward the median line. —*Copy.*

82—Woodhouse, W. B. The gingival trough —its early development, in: Australian Dental Journal xxxiii: 139-146. May, 1929.

This author's views are of interest as not being in full accord with those of Gottlieb. He traces the rôle of the enamel organ in the formation of the gingival epithelium through the generally accepted essentials of its development and notes that "the deep-

est part of the epithelial downgrowth maintains a constant position in relation to the floor of the nose or the lower border of the mandible." But, the question "whether accompanying growth—which must necessarily take place in this epithelium—is accounted for by proliferation of cells in its deepest layer, or any other particular position" he leaves undetermined—no sections having been found to show the actual cell division.

He describes the gradual shrinkage and epithelial replacements of the enamel organ and the initial trough formation, by the splitting of this epithelium frequently at a considerable distance from the tooth cuticle. With the deepening of the trough, the epithelial cells adherent to Nasmyth's membrane atrophy and are desquamated; thus obliterating the evidence as to the original production of the trough by a splitting in the cells themselves.

The deepening of the trough and the downgrowth of the epithelium usually progress *pari passu*. This certainly prevents one, in the opinion of the author, from accepting the idea of a continuous process of migration of the epithelial attachment toward the apex.

The author after showing very clear and interesting microphotographs draws the following conclusions:—

1. At the time of eruption, the epithelium covering the enamel is derived from the external epithelium and stratum intermedium of the enamel organ. As eruption proceeds, this epithelium seems to be reinforced by a downgrowth from the gum and with the formation of the pocket tends to replace this.

2. Attachment of the epithelium to the enamel surfaces exists through a membrane —the atrophied remains of the enamel organ which have become hyalinized.

3. At eruption the epithelium lining the enamel terminates at the amelo-cemental junction; but, in almost every case, with the formation of the pocket, the epithelium tends to grow down along the cementum in a thin column.

4. On eruption, there is no gingival trough. A trough is developed as the result of some stimuli other than growth; and is brought about by a splitting in the gingival epithelium itself, and not by a mere separation of the epithelium from the tooth cuticle. —*H. A. R.*

(b)—Periodontal Bacteriology

1927

1—Appleton, J. L. T. Jr., B. S., D. D. S. **The effect of rinsing the mouth and brushing the teeth upon the number of bacteria present in subsequent rinsings,** in: D. Cosmos. Oct., 1927.

Appleton attempts to study quantitatively the effects of (a) rinsing the mouth, and (b) brushing the teeth with a toothpaste, upon the number of bacteria in subsequent mouth rinsings. Numerous tests were made on three individuals, and the following conclusions reached: (1) vigorous rinsing of the mouth with tap-water produces on the average at most but a slight and transient decrease in the number of bacteria removed by an immediate second rinsing, and this decrease disappears within a little over thirty minutes.

(2) A second rinsing, which has been immediately preceded by a vigorous rinsing and by a thorough brushing with a simple, non-medicated toothpaste, contains on the average about 45 per cent less bacteria than the first rinsing. When the second rinsing is postponed until about sixty-five or seventy minutes after the first, the number of bacteria in the second approximately equals that in the first rinsing. When the second rinsing is made an hour and a half after the first, the number of bacteria in the second is 10 or 12 per cent greater than that in the first. These are average figures, but individual sets of rinsings show wide variations. (3) The results given in (1) and (2) seem to hold, irrespective of the absolute number of bacteria obtained in rinsings from different individuals. (4) It is noted (a) that the immediate decrease occasioned by brushing is much greater than that occasioned by simple rinsing, and (b) that while after simple rinsing the number of bacteria in the mouth removable by rinsing has come back within slightly more than half an hour to the base level of 100, this level is not reached after brushing, until slightly more than an hour has passed.

—*C. R.*

2—Beckwith, T. D., Ph. D., G. W. Simonton, D. D. S. and Rose, E. J. M. A. **The presence of bacterial organisms in human gingival tissue in gingivitis,** in: D. Cosmos—lxix: 164-171, 1927.

The authors demonstrated by a microphotograph the presence of bacteria along a lymph vessel in the submucosa of the gingiva in a case of gingivitis. In order to determine what organisms were present and viable they sterilized the surface with alkaline alcoholic extract of gentian-violet and brilliant green, injected ½ cc. of sterile physiologic salt solution into the gingiva and then aspirated one or two drops for culturing. Tincture of iodine was effective in sterilizing the surface but too irritating. Streptococci were present in the gingiva of all the 24 gingivitis cases examined, staphylococcus albus in fourteen and staphylococcus aureus in one. Different varities of streptococci were isolated including pyogenes (4), equinus (2), angiosus (6), salivarius (11), nutis (9), infrequens (2), ignavus (5), non-hemolyticus (2), equi (1), micrococcus gasogenes (1). Of eight cases showing no outward symptoms of gingivitis, seven were sterile and one, which showed radiographic alveoloclasia gave a positive pure staphylococcus albus culture. Repeated tests on the same men gave practically the same organisms each time. A large bibliography is given.

—*H. J. L.*

3—Fisher, John H. **Pyorrhea Alveolaris. Rôle of certain micro-organisms found in the lesions,** in: Amer. J. Path.—Mar., 1927; Abs. D. Cosmos—Aug., 1927.

This investigation was undertaken to study the probable significance of Endameba gingivalis, spirochetes, fusiform bacilli and streptococci found in the lesions of pyorrhea alveolaris. Direct smears were examined in 201 selected cases of pyorrhea alveolaris and in 17 control cases showing normal-appearing gums and teeth free from pyorrhea. Cultures were made from 30 cases of pyorrhea and also from the 17 control cases.

Endameba gingivalis was present in direct smears in 95.5% of 201 pyorrhea cases examined. Evidence is submitted that this ameba is a harmless parasite. It was not found in the clean normal gums examined.

B. fusiformis and spirilla were found in

the smears from all the cases of pyorrhea alveolaris studied and were also present in each of 17 normal gums. In the opinion of the author they are probably of little significance in the production of the pyorrhea lesion. Streptococcus viridans was present in each of 30 cases of pyorrhea alveolaris examined culturally. In all instances it was the predominating organism present on the blood-agar plates. In 17 normal gums, this organism was absent in eleven. Author decides that it is of much importance in pyorrhea.

Hemolytic streptococci were not present in any of the cases examined culturally nor were hemolytic influenza bacilli.

The pathogenicity of different strains of S. viridans isolated from pyorrhea lesions was tested upon rabbits with varying results. Some strains were apparently non-pathogenic; others killed the animals within 24 hours. One strain of S. mitis produced a fatal vegetative endocarditis, strikingly similar to that found in human cases dying of S. viridans endocarditis.

In a number of cases the blood serum of patients showing well-marked pyorrhea lesions, contained agglutinins for the predominating strain of S. viridans found in their lesions. (Abstr. of rev. in Cosmos.)

4—Graham, J. W. E. **The bacteriology of pyorrhea alveolaris,** in: J. M. A. So. Africa—I: June, 1927.

The author first considers the etiology, then the pathology of pyorrhea (which he thinks more properly named "chronic periodontitis") and in spite of his introductory promise to confine himself to pyorrhea, he goes on to discuss the "dead tooth." As to etiology he quotes Mummery and Broderick; while he notes from his own practice, cases of severe gingivitis in nursing mothers. This he ascribes to Ca insufficiency, which, he says, conflicts wth Broderick's theory; "but there is no theory adapted to all cases, which makes the study of pyorrhea so interesting"! In support of this argument he describes a case which he has treated for five years; "but the case gets steadily worse."

—*H. A. R.*

5—Koch, Dorothy A. **Relation of moisture and temperature to the viability of Endameba Gingivalis (Gros) in vitro,** in: Univ. of California Publications in Zool.—xxxi: 1927; Abs. D. Cosmos—Dec., 1928.

The work was done on cultures of this organism on artificial media. The thermal death point was found to 52° C. Immersion in water at 60° C. or above is a rapid and convenient method of sterilizing against E. gingivalis. The most minute quantities of liquid or solid material carrying amebae, if moist, may be infectious. From this it may be concluded that E. gingivalis may be spread both by direct contact and by an intermediate contaminated article.

—*Copy.*

6—McDonagh, Andrew J. **Study of bacteria in living pulps in relation to grinding teeth in periodontia,** in: Dominion Dental Journal—xxxix (8), 1927.

Author shows by a study of bacteriologic tests of a series of vital pulps that 65% of teeth which showed no break in enamel or cementum and were undoubtedly vital showed micro-organisms. Streptococcus viridans in most cases; staphylococcus aureus in 1/3 of the cases, and Bacillus coli communis in one case of a patient suffering from an appendiceal abscess. —*H. J. L.*

7—Peabody, W. A. Lewis L. C. and Hall, R. C. **Bacterial growth as a factor in the deposition of Calcium from saliva,** in: D. Cosmos—lxix (11), Nov. 1927.

This work continues that of Dr. Ivan Hall and collab. in the study of bacterial factors in pyorrhea alveolaris. Etiology is not discussed here; but the probable relation of bacterial growth to calcium deposition.

The initial and subsequent changes of mixed saliva *in vitro* are outlined here. Loss of Co2 produces an alkaline reaction in a few minutes. Incubation at 37° C. in 12-24 hours produces:

1. Loss of mucoid consistency;
2. Lessened opalescence;
3. Appearance of white sediment, sour odor and low pH, 6.6.

The acid reaction is due to carbohydrate fermentation from food and mucin; later there is a rise of pH which may reach a maximum of 8.6 in 48 hours, due to bacterial putrefaction, mainly of squamous epithelium and proteins.

Hall used saliva from two healthy persons

and demonstrated other significant facts. Later Hall and Westbay confirmed above results on samples from 21 persons. The object of the present study was to repeat part of their work, to determine the effects of incubation with excess fermentable carbohydrate on the Ca, and to demonstrate changes in sterilized saliva with pure cultures on Ca content.

The authors describe their methods and experiments, and reach the following conclusions:

(1) Ca determination by direct precipitation yields at least 93% of total Ca.

(2) Results of Hall and Westbay have been confirmed on changes in pH and dissolved Ca of incubating salivas.

(3) In addition, in contrast to rapid loss of soluble Ca from alkaline samples, there is no loss but rather an increase in soluble Ca. with development of acidity.

(4) Autoclaved saliva containing added soluble Ca, inoculated with fresh saliva shows similar results but at a slower rate.

(5) Further work on the problem should investigate comparative Calcium precipitating powers (extent and pH) of various streptococci. actinomyces, leptothrix and M. gazogenes. —*H. A. R.*

8—Strahl, H. Contribution to the flora of the root canals of pulpless teeth, in: Zahnärztliche Rundchau — Jan. 1927; Abs. D. Cosmos—lxix: 1075, 1927.

The bacteriological investigations of the roots of sixty periodontitic teeth and teeth with granulomata (40 under aerobic and 20 under both aerobic and anaerobic conditions) have yielded in 59 of the teeth, bacteria of the most diverse sorts.

Streptococci were the most frequent yield (80%); then staphyloc. (38.3%) then rods (35%) and cocci (16.6%). The 66 strains of streptococci isolated were differentiated according to their growth in different media, and by inoculation into mice. The pathogenicity was not determined. The hemolytic action in the form of growth on blood agar proved to be the most useful way of bringing out distinctions. In this way the streptococci were classified as follows:

S. hemolyticus and similar 27.2%
S. viridans and similar 22.7%
S. mucosus 7.5%
S. putrificus were found 4 times
Not classified 34.8%
—*Copy.*

9—Thomson, M. J. Streptococci found in pyorrhea alveolaris, in: Edin. Med. Jour.; Abs. Austr. Dent. Summary—vii (9), 1927.

Three types were observed: S. mitis, S. fecalis and S. salivarius. The virulence of the streptococci was experimentally studied on animals. Arthritic changes were absent entirely in all cases with one exception where the streptococci was of hemolytic type and came from a case of osteomyelitis. Endocarditis could be produced by the injection of streptococci alone, and in most cases it was due to the S. salivarius. Kidney lesions especially of interstitial type were observed, and the streptococci could be recovered from the urine. The only suppurative lesions found affected kidneys and heart; in all cases these focal infections were to be referred to the S. salivarius, whence it appears that this organism is more likely to become localized and produce pus than S. fecalis. By the injection of streptococci, recovered from cases of oral sepsis, choreiform movements were induced. —*Copy.*

1928

10—Burdon, K. L. B. melaninogenicum from normal and pathologic tissues, in: J. Inf. Diseases. Feb., 1928; Abs., D. Cosmos—lxx, June, 1928.

This organism (see D. Cosmos, July, 1921, p. 750) constantly inhabits healthy mucous membranes but takes a prominent part in various pathologic processes. It occurs constantly and often in large numbers, in the normal human mouth, being chiefly localized on the gum margin, and also in the mouths of guinea pigs, rabbits and dogs, in the crypts of the tonsils, and on the male and female genitalia. This organism probably acts as a secondary invader and does not necessarily have any primary etiological significance; but in many cases of lung abscess, pyorrhea alveolaris and puerperal infection it occurs in such large numbers that it must constitute an important bacterial factor in these diseases. In pyorrhea alveolaris the abundance of growth from about the affected teeth increases definitely with the progress of the lesions and in many cases the

organism becomes predominant in advanced stages of the disease. However, this may only be because the conditions become more and more favorable for its development.

—*Copy.*

11—Ferrier, E. Researches on the bacteria in the human mouth and their localization, in: Rev. Mens. Suisse d'Odontologie—Apr., 1928; Brit. D. J. xlix: Dec. 1928.

This is an exhaustive study of the bacterial flora of the mouth based upon direct examination from a large number of mouths. The organisms present at the cervical margin, in carious cavities and the flora of the tonsil were all examined and an attempt made to correlate the findings at these three sites. There was a close but not identical similarity between the types of bacteria and their order of frequency at these sites. Tables are given showing findings in clean and dirty mouths.

The paper does not lend itself to summarizing, but the author's conclusions are as follows: the bacterial fauna and flora of the mouth and in carious teeth are extremely various and inconstant. It is not possible to find a definite order of frequency and abundance characteristic for caries, the gum margin, and the tonsils. The leptothrix is constantly present on the gums, the spirochaetes are particularly numerous at the gum margins, etc.

The lack of oral hygiene does not appear to exert any considerable influence on the bacterial flora; but there is a distinct increase in frequency of spirochaetes at the gum margins. —*Copy.*

12—Hartzell, Thomas B. M. D., D. M. D., Bacteria, in: J. Am. Dent. A.—Mar., 1928.

The author states, "I can truthfully say that I have never found a lesion in the soft tissue, nor in the hard tissue of tooth structure, nor in the dental pulp, except only the traumatic lesion, which did not trace its origin to the action of bacteria, either as the result of the action of their waste material or of their digestive ferments. To those who follow the profession of dentistry, who grasp this fundamental fact and create and carry on opposition to the destructive forces of bacterial life most efficiently, will come the greatest measure of success obtainable."

After a brief statement of the usual methods of immunizing living bodies Hartzell tells of Larson's "more recent discovery of a method to render all bacterial poisons harmless without causing them to lose their power to call out anti-bodies, thus making it possible to administer enormous doses of toxin without danger and thereby producing rapid immunity to bacterial poisons."

The author claims we can now "produce local immunity with the detoxified living pyogenic organisms found in vast numbers on surfaces of roots of teeth in deep pyorrhea pockets—frequently five hundreds layers deep. These can be reached by sodium ricinoleate in the form of a two per cent liquid or a paste pressed around the teeth by tooth brush bristles, then a vigorous massage to drive the detoxified bacteria and their poisons directly into the pocket thus vaccinating it." "The living germ in its most active state at the site of growth, but robbed of its power to destroy or poison tissue cells while losing none of its power to promote the formation of anti-bodies, is thus pressed into the bleeding pyorrheal ulcer." —*C. R.*

13—Hinshaw, H. Corwin. Experimental infection of dogs with endamoeba gingivalis and trichomonas buccalis of human mouth, in: Proc. Soc. Exper. Biol. and Med.—March, 1928; Abs. D. Cosmos—lxx, July, 1928.

Hinshaw's first success was with an old dog with an advanced gingivitis. Several weeks of careful observation demonstrated that there was no spontaneous infection. A culture containing E. gingivalis and trichomonas b. recently obtained from a case of human active pyorrhea was inoculated subgingivally. Both species continued to multiply in the dog until he was sacrificed 14½ months later. Striking pathological changes were observed closely simulating human pyorrhea. The greatly accelerated rate of deposition of dental calculus was most marked. This experiment was quite inconclusive as to etiology of the lesions because of inadequate controls.

More recently Hinshaw successfully infected four other dogs with E. gingivalis after definitely demonstrating the absence of protozoa before inoculation. In every case there was more or less inflammation and gin-

gival pocket formation prior to the beginning of the experiment. Only such dogs were susceptible. These and the controls were carefully kept under optimum conditions with properly balanced diets. None of these have borne the infection longer than 4 months. No gross pathologic change has been observed to date and the excellent environmental factors have a systemic improvement in most cases, which is reflected in the oral condition. —*Copy.*

14—Hinshaw, H. C. and F. V. Simonton. **The amebas of the mouth in relation to incidence of pyorrhea; clinical symptoms, extent of lesions and age of patients,** in: J. Dent. Res.—Aug. 1928; Abs. Cosmos—lxxi: Mar., 1929.

The incidence of infection with Endameba g. in 357 individuals examined was 46.34%. These parasites do not occur in the strictly normal mouth. They are invariably found in typical inflammatory pyorrhea alveolaris from an early stage of the disease until its termination. Their presence is invariably associated with the existence and extension of gingival pockets. They are more commonly found when the gum margin has receded. 57 per cent. Microscopic pus is always demonstrable in cases of amebic infection of the mouth. Hypertrophy of the gum may occur, but is not an invariable accompaniment of amebic infection. Supragingival calculus is always present.

Subgingival calculus cannot always be demonstrated in early cases of amebic infection especially when the teeth are frequently scaled. In later stages of pyorrhea it is always present.

Endameba gingivalis is less likely to occur in mouths with moderate amounts of detritus than in those with slight detritus, provided everything else is equal. This argues against the fact that it is a saprozoön feeding largely upon bacteria and food debris. Conditions of the saliva associated with mild caries do not appreciably affect the ameba of the mouth. Nor is there any demonstrable association between a history of caries and predisposition to amebic infection. The inflammatory reactions associated wth amebic infection are in the nature of a chronic process. Certain acute localized inflammatory processes do not support E. gingivalis.

Persistent and intelligent efforts of the patient to secure good mouth hygiene are not effective in destroying this parasite.

The saliva of amebic hosts is usually either plentiful or of a consistency that forms a glairy film on mucous surfaces.

E. gingivalis thrives on a concentration of neosalvarsan and mercury that is lethal to Treponema pallidum.

The incidence of infection with the ameba of the mouth is very much higher in persons belonging to higher age groups. The incidence of infection was found to be 26.74%, 51.05% and 71.06% respectively, in the four quartile groups arranged from youngest to oldest. —*Copy of abs.*

15—Howitt, B. F. Fleming, W. C. and Simonton, F. V. **Study of effects of various diets on hygiene and microbiology of mouth without and with use of toothbrush,** in: Dental Cosmos—lxx, June, 1928.

These authors have attempted to show the effects of various diets on the growth of bacteria on the tooth surfaces. An inmate of San Quentin prison with a normal set of teeth and healthy gingiva was selected. After thorough examination and recording his teeth were cleaned, and he was placed on an experimental diet for a week after which the mouth was photographed, bacterial smears and cultures made, the mouth again cleaned and another diet started. The diets were selected primarily for differences in detergent effects on the teeth and were first used without the toothbrush and later repeated using the toothbrush. For one week the patient was fed through a stomach tube, in order to note the effect of lack of food débris and lack of masticatory function. The authors' conclusions follow:—

(1) "Desquamated epithelium constitutes an important element of the oral débris upon which, with addition of salivary constituents, the usual mouth organisms flourish as abundantly as they do upon débris gathered following any of the diets used.

(2) "Débris gathered following the 'sticky' diet showed no more abundant growth of organisms than did that following the supposedly detergent diets except that there was a slight increase of aciduric forms on the carbohydrate diet.

(3) "Artificial cleansing with the toothbrush is many more times effective in reduc-

ing the absolute numbers of mouth organisms than is the supposed cleansing accomplished through a diet composed presumably of detergent elements.

(4) "When the toothbrush was not used, the appearance of the teeth was always cleaner following the 'sticky,' 'main line' and balanced diets than on the supposedly detergent diets. When the toothbrush was used, the appearance in each case was similar."

The report shows painstaking care and is a valuable contribution to our knowledge in this field. —*H. J. L.*

16—Keilty, Robert A. **Present state of our knowledge of gingivitis,** in: J. Lab. and Clin. Med.—xiii: 451-468. 1928.

Interesting article from a bacteriological standpoint. Author studied 5000 cases bacteriologically; finds spironema in 96.4%, Endameba in 71.6%, and definite bacterial flora in 66.6%; thinks the spironema and fusiformis are forms of a leptothrix; that treatment should be based on etiologic microorganisms; that scaling should be discarded; that the term gingivitis should include all forms of periodontoclasia; claims the H-ion concentration of the gingival exudate is usually greater than that of saliva and may reach pH 4. —*H. J. L.*

17—Lazarus-Barlow. P. **A bacteriological examination of the alveolar bone in relation to pyorrhea,** in: Brit. Dent. Jnl.—Jan., 1928; Abst. Dental Cosmos—lxx: June, 1928.

(1) Out of 516 cultures taken from the alveolar bone of 172 subjects in the postmortem room, 424 (82.17%) were sterile.

(2) The 92 strains isolated were from 54 subjects.

(3) The organisms most frequently isolated were streptococci (57 strains) and staphylococci (20 strains).

(4) Experiments to try and correlate the organisms found in the alveolar bone and those from the knee-joint in arthritic conditions were mostly negative.

(5) Animal experiments failed to produce lesions suggestive of pyorrhea alveolaris. —*Copy.*

18—Roberts, George Fenn, M. D. **Ptyalin-Blood Broth: A new medium for the cultivation of mouth organisms,** in: J. Am. Dent. A.—Aug., 1928.

The object was to make a fluid that would represent the saliva as found in the ordinary mouth. Many organisms found in mouth smears refuse to grow on the commonly used medium. The material required for the new medium is as follows:

	C. C.
Flaked Corn (maize)	10.00
Distilled water	1,000.00
Dessicated parotid gland	1.00
Dessicated submaxillary gland	1.00
Dessicated sublingual gland	0.50
English drop chalk ($CaCO_3$)	10.00
Defibrinated Sheep's blood	100.00

Soak the flaked corn in the distilled water overnight. Then boil until corn is well cooked and pass through a tomato sieve. Allow to cool and add the glands and the chalk using glass pestle and mortar to mix them. Place in an incubator and shake from time to time. Next morning test with iodine. There should be no violet coloration. Then add the fibrinated blood and about 5.00 C. C. of toluene. Incubate again for forty-eight hours. Boil the mixture for about twenty minutes and strain through cheese cloth and absorbent cotton pouring enough distilled water over the strainer to keep 1 liter. Measure out 10.00 C. C. and drop in lime water to make the pH 7.5. Then add enough lime water to bring the whole amount to this reaction using brom-thymol blue as the indicator. Sterilize. The mixture should be perfectly clear and of a light straw color. Streptococci grow especially well in this broth. Used in root-canal clinical work in the Dental Department of the University of Buffalo. —*M. M. B.*

1929

19—Antoniotti, Dante. **Ueber Kultur der strenganaeroben Mikro-organismen der Alveolarpyorrhöe,** in: Revista odontologica v: 331-351. 1929; Abs. D. M. f. Z.—xlviii (4), 1930.

From the pus of pyorrhea the author isolated the following anaerobic microbes:

1. the familiar B. fusiformis.

2. an anaerbic, nonhemolytic streptococcus, giving off a fetid odor in cultures.

3. one resembling the gas gangrene bacillus with proteolytic qualities, Bacillus H.

4. one resembling the diphtheria bacillus, Bacillus V.

5. a Bacillus Z which evolves acid.

6. a Bacillus building heaps or mounds in the culture, Bacillus X.

(*Brief abstract of German review of Italian article.*) —*H. A. R.*

20—Beckwith, T. D., with A. Williams, and E. Rose. The role of bacteria in pyorrhea, in: NY.—Med. Jour. & Record—129: Mar. and Apr., 1929.

This research was carried on with the original personnel of the California Stomatological Research Group, and experiments were carried, some on rabbits and guinea pigs, some on men in the San Quentin Penitentiary. There are two factors in the development of any infectious disease.

(1) an infectious agent which gains access to the tissues; and

(2) susceptibility to infection on the part of the tissues.

Knowledge of tissue resistance is exceedingly limited. The authors review the numerous writers on the various infective agents at work including protozoa, particularly the amoebae. The very complexity of possible bacterial etiology causes doubt that any single form is to be considered guilty. Nevertheless, as stated by Appleton, "The existence of an infectious agent must be granted." Previous work of these authors has shown that there is a portal of entry into the human gingivus (sic) in cases of pyorrhea. 161 sets of cultural tests were made on a group of 24 men with gingivitis. Cultural isolations from these 161 aspirations from gingival areas, are tabulated. From every case of human gingivitis examined it was possible to demonstrate the presence of living bacterial organisms by culture. With the use of pyorrheal pocket contents from frankly active cases injected into the peridental membrane of rabbits and guinea pigs through the alveolar bone, all attempts to transmit the condition were failures. Men from the San Quentin prison were then treated with autogenous bacterins, the suspension for each individual being composed of the complex of organisms originally isolated from himself. Improvement occurred in all save one of the cases cited.

The authors realize that their series are too small for definite conclusions; yet the results are suggestive. "An autogenous dead antigen replaced locally within the area of original isolation depressed temporarily at least, the activity of infection."

In further discussion, the authors state that there are factors controlling susceptibility within the human host; they do not know what these are; nor have they any proof that bacteria play other than a secondary role.

Their conclusion is, that bacterial infection is of importance; that its activity may be depressed by local immunological technic; that regeneration of peridental soft tissues commences so that the teeth become more firmly set; and that, even though improvement may not be permanent, it shows that infection is an important contributing factor in pyorrhea. —*H. A. R.*

21—Cook, Thomas J., D. D. S., and Stafne, Edward C., D. D. S. Bacteriologic study of pyorrhea alveolaris, in: D. Cosmos—lxxi:, Feb., 1929.

This is a study of the relations between alveolar pyorrhea and metastatic infections. From deep pyorrhea pockets the streptococcus was cultivated, rabbits being used as experimental animals. Those receiving injections of streptococci from the patients with alveolar pyorrhea and arthritis, showed arthritic symptoms in 30% of the cases. Under like circumstances animals injected with cultures from patients with pyorrhea alveolaris and iritis showed 37% iritis; from those with alveolar pyorrhea and colitis, 25%; ulcerations of stomach and duodenum, 42% upward. When cultures were used from patients who had only alveolar pyorrhea, the percentages were significantly smaller.

(*Transl. of review in D. M. f. Z.*) —*H. A. R.*

22—Greenberg, Samuel, D. D. S. and Greenberg, Philip, D. D. S. The incidence of Spirochete and Fusiform Bacillus in gingival inflammations and its diagnostic value, in: Dental Cosmos—lxxi, April, 1929.

Of two hundred and fourteen smears taken from unhealthy gum tissue, one hundred and eighty-nine or 89 per cent showed spirochetes. Stafne's studies of smears from

two hundred and fifty cases showed that the longest spirochetes were found in the unhealthiest mouths. He found the fusiform bacillus in 57 per cent of those cases containing spirochetes. Of one hundred and nine smears taken from apparently healthy gingivae, seventy-nine or 72 per cent were free of spirochetes. The rest had spirochetes in varying quantities, but only one showed "many" (twenty or thirty). Of the smears from healthy gingivae 50 per cent also contained fusiform bacilli in different amounts. Fifty per cent of the smears from unhealthy gums showing the spirochete also contained the fusiform bacillus. Smears taken from cases which represented the typical clinical picture of Vincent's infection, had no more spirochetes than the average case of pyorrhea. As to the fusiform bacilli, very few smears showed large numbers and many smears showed none. Foul breath seems to be considered one of the important factors in the recognition of Vincent's infection but the fact of the matter is that in our work we have found that many patients whose gums have "countless" numbers of fusiform bacilli have no bad breath at all. Large doses of fusiform bacilli injected into animals for serological tests produced no serious results at all. They are probably scavengers of disease and not particularly harmful. The cause of Vincent's infection must remain a matter for further research until the disease has been produced in susceptible animals by the spirochetes and fusiform bacillus.

—*M. M. B.*

23—Grosset, W., Ass't. at the State Institute of Stomatology and Odontology at Moscow, U. S. S. R., Director, Prof. Dr. Eudokinmoff. **Étude de la flore buccale,** in: Revue de Stomatologie,—xxxi, April, 1929.

For three years this study has been carried on, concerning the contents of the foci of infection, viewed bacterioscopically, in pyorrhea alveolaris.

Grosset divides the bacterial flora of the buccal cavity into three classes:

A—Those constantly present—

1. Leptothrix buccalis.
2. Bacillus maximus buccalis.
3. The group of cocci.
4. The group of bacilli.
5. The association of fuso-spirillary b. (he includes here the spirochetes).
6. The protozoa.
7. The virus filtrans.

B—Variables—

The microbes of the air, water, and foods: B. mesentericus, B. coli communis, the staphylococci, yeasts, mycetes, and sarcinae.

C—Accidental—

B. of Koch, Loeffler's bacillus, actinomyces, and others.

Transl.—H. A. R.

24—Rickert, U. G. **Streptococci as I know them,** in: J. A. D. A. xvi. 840, 1929.

The author deals with the problem of periapical infections and the part which streptococci play in their etiology. Emphasis is given to the initial changes that render the periapical tissues susceptible to infection, such as trauma, potent drugs and diffusion of foreign toxic substances into the periapical circulation, conditions which usually precede infection. Patients who can build up a defensive quarantine in the form of a granuloma are as a rule less apt to be disturbed by these infections and are less susceptible to secondary infections. There appears to be two possible types of periapical changes due to intoxication in the absence of infection: First, proteolytic changes where pulps have been removed without injury to the periapical tissues; and secondly, those resulting from instrumental and chemical trauma. It is believed that degenerative changes in the periapical region "frequently precede actual infection of the involved parts." Powerful drugs are a potent factor in lowering the resistance of the periapical tissues. These toxins have as yet never been isolated and demonstrated chemically, serologically or immunologically; the evidence of their part in periapical infections rests on a combination of clinical and laboratory experience.

In the treatment of pulpless teeth two procedures are outlined: First, those cases in which it is the part of wisdom to extract all pulpless teeth; Secondly, those in which certain fundamental conditions can be met and the teeth retained. It is recommended, however, that "in all cases of pulp involvement, if the tooth can be dispensed with

without serious inconvenience and loss of function to the patient, and a restoration will not be required for the lost member, extraction is justifiable." A note of warning is sounded regarding "pulp involvements that have too frequently followed the extensive preparations of the supporting teeth," (for restorations) which have been proven to be the initiating factors in certain secondary infections that are quite as serious as are those from pulpless teeth. Attention is called to a preparation known as "root sealer" compounded by the author which is believed to meet many of the requirements of a root canal filling. The article concludes with the statement that "if we as a profession are to solve the problem sanely, we cannot afford to become involved in extraction hysteria, because such measures fail to lead us forward." —*A. H. M.*

25—Thibault and Raison. **Septicémies d'origine bucco-dentaire, pyorrhea alveolaris, gingivite interstitielle pericoronaritis suppurée. Les microbes de la pyorrhée alvéolaire,** in: Rev. de Stomatologie—xxxi (9), Sept., 1929.

Pyorrhea alveolaris constitutes in reality a mixed focus of infection, ready at once to pour its septic products into the mouth as in all gingival suppurations and to eliminate them by the blood or lymphatic paths at the summit of the alveolar walls. The disease is characterized by a progressive destruction of the ligamentary tissues due to the pathogenic germs of the mouth, and through a simultaneous resorption of the alveolar edges favored by different constitutional states. The infection starts at the periphery of the tooth, lighted up by all the irritations and chronic inflammatory conditions of the gums. After the epithelial investment is passed, the ligaments become infiltrated and destroyed; the microbic propagation into the depths of the tissues is facilitated by the lymph and blood vessels which run toward the apex parallel to the root.

The author then describes the formation of the pocket, the epithelium lining the periodontal space with its breaches, through which the microbes invade the fibro-granulomatous tissue, the vessels on the ulcerated walls, and the adjacent bones.

The drainage difficulties of these narrow pockets, inaccessible to the cleansing influence of the saliva, and the consequent stagnation of septic products are easily conceived; but less easily appreciated is the extent of the suppurating surface. The author here cites the supposition of J. Rodway (Brit. Med. J., Mar., 1926) that "if the different alveolodental articulations could be unrolled and laid side by side, in a case of pyorrhea of medium intensity, one would get a surface such as could be covered with the palm. Then we can realize that this extent of surface conceals a pus pocket which can be only imperfectly drained. Of course, it may be objected that in certain forms of polyarthritis, the suppuration drains freely into the oral cavity; but we must not overlook that even in this case, the germs swallowed may be absorbed by the intestinal capillaries and enter the general circulation by way of the portal system."

The authors discuss the bacterial flora. Brown and Coxon are quoted. Their statistics indicate that pyorrhea is always a mixed infection with staphylococcus and streptococcus predominating. Only Weston A. Price admits the possibility of spirochetal septicemia; but it appears these are rather responsible for certain pulmonary infections, transmitted by way of the respiratory passages. —*H. A. R.*

26—von Beust, Theo. B. **Morphology and growth of fusiform organisms,** in: J. Am. Dent. A.—xvi: Apr., 1929.

"The student of the fusiform bacillus would probably do well to dismiss for the present the thought that fusiform organisms are bacteria. Rather a generation of spores should be imagined, developed from a parent which has affinities with the aquatic fungi." As to the identity of the spores of Leptothrix falciformis and the fusiform bacillus, the author thinks this is a still open question. However the granules or spores shown in his well-illustrated article, together with the central threads must be recognized, he thinks, as organic units. He concludes that however the question of affiliation of these forms may be decided finally, it is a fact that the peculiar type of schizogenesis observed in the propagation of the fusiform bacillus removes this form from the schizomycetes. —*H. A. R.*

(c)—Other Laboratory Researches
(Dietetics, Physiological Chemistry, Etc.)
In Relation to Periodontology

1927

1—Hirschfeld, I., D. D. S. **Interdental canals,** in: J. Am. Dent. A., 1927: Abs. D. Cosmos, lxix: June, 1927.

Author's summary:—

(1) The linear and radiolucent circular areas described in this paper are produced by interdental canals and their foramina, respectively, which are normal anatomic features and not the result of pathologic bone destruction.

(2) They may be mistaken for evidence of periodontal disease in the same way as are some of the other radiolucent markings.

(3) A knowledge of their anatomic characteristics may serve as an important aid in diagnosis.

(4) In certain cases of doubtful periapical or parietal radiolucent areas in the lower incisal region, it is advisable to take additional radiograms at different angles for the purpose of ruling out anatomic features.

(5) The function of the interdental canals and foramina is to transmit well-sized blood vessels through which the inferior dental and sublingual arteries anastomose.

(6) The constancy with which the foramina appear in the mandibles not only of man, but also of many other mammals, and in approximately the same relative position, suggests their functional importance.

—*Copy.*

2—Ketcham, Albert H., D. D. S. **A radiographic study of orthodontic tooth movement:** A preliminary report, in: J. A. D. A., xiv. Sept., 1927.

A radiographic survey of 385 treated orthodontic cases shows definite resorptions of apical root ends of some of the anterior teeth of eighty-two patients or 21 per cent. The maxillary anterior teeth are more disposed to resorption than the mandibular. These cases are true resorption and not anatomic anomalies. Radiograms made at the beginning of treatment show that there was normal root length, while those made at the conclusion show that resorption has occurred. Careful consideration brings the conclusion that there is something besides appliances involved in this problem. It must be concluded that some people are more susceptible to disturbances during tooth movement than others. There may be a hitherto unrecognized susceptibility to pathologic effects from mechanical stimuli. The poorly balanced diet of the majority of our orthodontic patients may be an underlying cause of resorption of tooth roots, namely, lack of lime salts or calcium starvation or faulty metabolism. The orthodontist must consider to what degree the appliance, through immobilization interferes with the normal function of the teeth and for how long a period this interference continues.

—*M. M. B.*

3—Marshall, John Albert, D. D. S., Ph. D. **Diet in relation to disease of the supporting tissues,** in: J. A. D. A., Dec., 1927.

Dr. Marshall concerns himself in the main with the general principles of cell metabolism.

The work of Howe. Mellanby, Grieves, and McCollum, he says, has traced a closer relationship between diet and teeth than has been supposed to exist; and in his own experiments on dogs, he has been able to produce, through unbalanced diets, changes in the supporting structures clinically almost identical with the earlier stages of pyorrhea. The pus stages and pocket formation he has not as yet been able to develop in his experimental animals.

If or when the laboratory worker can produce these pyorrheal manifestations by means of malnutrition and faulty diet he will establish the disease as a local manifestation of a general disorder.

The ultimate correction then will come from first—a local prophylactic treatment or deep scaling, the correction of occlusal trauma and elimination of infective pro-

cesses; second—a carefully devised systemic treatment based on a knowledge of foods and metabolism.

Finally he would emphasize the fact that the beginning of digestion is in the mouth, and diet and digestion should be understood by the dentist whose province it is to restore the masticatory mechanism to normal function. —*C. R.*

1928

4—Glasscheib, A. and Honsig, **Zur Pathogenese und Therapie der Alveolarpyorrhoe,** in: Ztschr. f. Stomat. xxvi, p. 440, 1928. (Preliminary communication.)

Author notes that:

Jacques (of Nancy) calls attention to the usual presence of alveolar pyorrhea in ozaena; and believing in a relation between the two, recommends a polyvalent vaccine for ozaena, also exhibited in pyorrhea.

These authors, regarding pyorrhea alveolaris as a product of A-avitaminosis, have instituted experiments with phosphorated cod liver oil.

5—James, W. Warwick, OBE., LDS., FRCS., LRCP., and Arthur Counsell, (pathologist). **The primary lesion in "so-called pyorrhea alveolaris,"** in: Brit. Dent. Jnl. xlix. Oct., 1928.

(General considerations summarized by the author.)

The primary lesion is local in origin, a statement founded upon a correlation of histological and clinical observations.

The disease is cured by extraction due to removal of the surface on which the deposit takes place.

"Pockets" regarded as characteristic of the disease are found to be completely occupied by calculus in the deepest aspects and lined by epithelium.

The teeth are affected in unequal degree, pointing to local causes, such as malposition of teeth, mouth-breathing, impaired function, or any factor forming deposit.

The interdental origin of disease is at a site where deposit readily occurs. Local treatment can effect such improvement that the disease may be arrested for years.

Complete control of the mouth-breathing, and local treatment may effect improvement lasting for years.

The association of calculus with progress of disease depends upon deposit in depth of pocket, and not upon gross collections readily seen.

The late inflammatory infiltration of the periodontal membrane is a remarkable contradiction of the view that this membrane is the main seat of disease. —*Copy.*

6—**Editorial.** British Dental Journal, xlix (20); Oct., 1928, re paper of James (W. Warwick) and Counsell (Arthur) in this issue.

The editorial writer compares the histological findings of James and Counsell, Gottlieb, and Box in regard to disputed points in periodontal histology. He emphasizes the agreement on the epithelial proliferation along the cementum in periodontoclasia and notes the points of disagreement in interpretation of the role of this epithelium in pocket formation.

A very interesting and valuable discussion of the points made in this paper is reported. —*H. J. L.*

7—Jones, Martha R., Ph. D., and F. V. Simonton, D. D. S. **Mineral metabolism in relation to alveolar atrophy in dogs,** in: J. A. D. A., xv (5) May, 1928.

The authors group the types of pyorrhea as follows:

1. Inflammatory—
   a. Local. b. Systemic.
2. Non-inflammatory—
   a. Local. b. Systemic.
3. Combinations of the above.

The non-inflammatory causes not including scurvy poisoning of various kinds are considered in this paper. A series of 138 puppies representing 25 litters were tested by various diets to determine the effects on bone and especially on the interdental alveolar process. Among other things it was discovered that great variation in the mineral content of bones exist in new-born puppies even of the same litter and apparent similarity in size and nutrition. The normal diet used consisted of bread, milk, meat, butter, fat and orange juice in carefully proportioned quantities ground together.

The Ca and P quantities were varied by adding salt mixtures high in one and low

in the other. The animals were weaned in four weeks and put on the basal diet for two weeks, after which urine and blood studies were made for retention of Ca and P and roentgenograms were taken of the bones.

The authors summarize the results of their experiments as follows:

1. The dog develops spontaneously skeletal and dental defects comparable with those seen in man, and for this reason is superior to the rat for certain metabolic studies.

2. Litters of puppies and even individuals of the same litter vary tremendously in their reaction to the same diet and hygiene, and in order to evaluate experimental results, a knowledge of the initial skeletal condition of the animal is of prime importance.

3. In the absence of sunshine, two factors are primarily concerned with the deposition and resorption of bone in puppies, the first and probably the more important being the metabolic state which is inherent in the individual; the second, diet.

4. An excess of alkali in the diet, regardless of its calcium-phosphorus ratio (within the limits used), consistently initiated retrograde bone changes in apparently normal puppies and greatly accelerated the destructive process in subnormal individuals. Concomitant with retrograde changes there was an increase in the excretion of bone salts.

5. The omission of the alkaline salt mixture from the diet of animals undergoing retrograde bone changes resulted in rapid and profound bone destruction in spite of the fact that the diet was well constituted.

6. Hydrochloric acid in quantities sufficient to neutralize the excess of alkali in the diet, cod-liver oil and roentgen rays all promoted calcification.

7. The seat of disturbance in faulty mineral metabolism appears to be in the bone forming cells rather than in the gastro-intestinal tract as has been generally believed.

8. Dental conditions of great interest have been found associated with skeletal defects produced on experimental diets and have been described.

9. Diets which are potentially basic in reaction, low in Ca, protein and vitamins (Bread, meat and potato) with and without the addition of sodium carbonate, have, in all cases so far examined, resulted in retrograde changes in the alveolar bone of healthy, robust adult dogs. These changes, namely halisteresis of the crests of the septal processes, osteoporosis and lacunar resorption of the roots of the teeth are strikingly similar to the diffuse atrophy in human tissue described by Gottlieb. These lesions are regarded as fundamental to the non-inflammatory type of parodontoclasia of systemic origin, the most destructive and hitherto obscure form of pyorrhea.

10. The substitution of rice for potato in the diet was accompanied by a regeneration of alveolar bone and remarkable reparative phenomena in the roots of the teeth. The substitution or repair of eroded dentin by haversian bone is described and to our knowledge for the first time.

11. Evidence is presented indicating that roentgen rays may be a therapeutic measure of great value in the treatment of parodontoclasia and an aid to the repair of bone.

12. The alveolar process appears to be more susceptible to slight dietary faults than do the long bones and in young puppies, responds more slowly to therapeutic measures.

13. A study of the food habits of races which are relatively immune to dental and skeletal disease shows that a high protein diet which is potentially acid in reaction (flesh) even though it is relatively low in Ca, is well suited to the needs of those inhabiting the arctic regions in spite of the fact that a large portion of their lives is spent in darkness. On the other hand, the Ca salts in a low protein, basic diet (fruits and vegetables) may be equally well utilized in those regions where sunshine is abundant.

14. Clinical observation and experimental findings indicate that ossification is the resultant of a large number of factors. It is believed that bone is a more labile tissue than it has generally been thought to be, and that ossification depends on the maintenance of a certain balance of ions between the bone cells and their surrounding medium. If this is true, any factor of any origin whatsoever, if it is capable of altering that balance either by action on the cells themselves or the tissue fluids, is a determining factor in that individual in regulating the deposition and resorption of bone.

The article is well illustrated and is an

important contribution to dietetic and periodontal research. —*H. J. L.*

8—Karlström, Sam, D. M. D., and Prof. Wm. J. Gies (Col. U., Biol. Chem. Lab.) **Does the excessive ingestion of milk favor deposition of salivary calculi?**, in: Jour. Dent. Res., viii, Abs. 20, Apr., 1928.

Milk contains or in its assimilation yields, a large proportion of earthy phosphate, the chief ingredient of salivary calculus. An effort has been made to determine whether the ingestion of milk in abundance, as often occurs under modern dietary recommendations, might be attended by increased formation of hard tartar.

The experiments which thus far have been preliminary only were restricted to analyses of saliva obtained from a man 29 years old. He was in good health, and had normal gums and no pyorrhea. Tendency to tartar formation was very slight and for several years there had been few new signs of dental caries. Three daily dietetic conditions were compared during a period of 51 days: (a) normal diet; (b) normal diet plus an extra liter of water; (c) normal diet plus an extra liter of milk. The effects of these conditions were compared on a daily routine that was uniform in all other respects. Suitable intervals and alternations prevented overlapping of effects. Specimens of saliva were obtained at the same relative periods and by mechanical stimulation (chewing pure paraffin.)

Familiar variations in composition similar to those of saliva on any physiological basis, were noted, but there was no significant increase in the content of Ca, P, or calcium phosphate. There were no indications of any increase in tartar formation. The senior author's plan includes extensions to apply to conditions at middle age, including persons in good health and others having different degrees of periodontoclasia. —*Copy.*

9—Karshan, Maxwell and Joseph Schroff. **Composition of some salivary calculi,** in: J. Dent. Res., viii: 454-5, 1928. Abs.

In an analysis of human salivary calculi from various sources, Ca, P & Mg were determined by the method of Howland, Marriott and Kramer; CO_2 from carbonate by method of Shear and Kramer. The distinctive features of these methods is the elimination of ashing, which, when organic matter is present (as pointed out by Wells) converts organic P into phosphate and C into carbonate, thus yielding erroneous figures for Ca, Mg. P and CO_2 if expressed as Ca phosphate, Mg phosphate and $CaCO_3$ —compounds presumably present in salivary calculi. The content of organic matter was determined by ashing and subtracting weight of ash from total. The material was extracted with a mixture of alcohol and ether in a Soxhlet apparatus for the separation of lipins. All calculations were made on a dry basis, the material having been heated to 100 C to constant weight before analysis.

This is followed by a table showing the analysis of various single calculi from the rt. and lft. submax. glands as well as groups of small calc.

After subtracting enough Ca from the total Ca to make calcium carbonate, and after taking sufficient P from the total P to make $Mg_3(PO_4)_2$, the ratio of residual Ca to residual P is successively 2-01, 2-05, 2.31, 1.90, 2.05 for samples, 1, 2, 3, 4, 5, resp. In $Ca_3(PO_4)_2$, the ratio of Ca to P is 1.94. The closeness of the above ratios to the theoretical 1.94, justifies the conclusion that the Ca phosph. in saliv. calc. exists as $Ca_3(PO_4)_2$. —*Copy.*

10—McCall, J. O., A. B., D. D. S. **Variations in the periodontal structures of erupting and recently erupted teeth, as disclosed by the roentgenogram,** in: Jour. Dent. Research, viii: June, 1928.

The intraoral radiogram properly interpreted may be useful as a guide to the nutritional state of the individual and may point the way to dietary or therapeutic measures needed for the general welfare. It may also enable the individual to build up an adequate resistance against periodontal disease. Variations from what seems to be normal are indicated by increased width of the pericemental line, increased width and density of the so-called lamina dura, and increased radiolucence of the circular zone over the root end. These variations are not always accompanied by variations in the mouth tissues discernible by local examination. —*M. M. B.*

11—Rygge, Johan. **Blood studies in cases of "paradental" inflammation,** in: Ztschr. f. Stomatologie, June, 1928. Abstr. D. Cosmos, lxx: 1035. Oct., 1928.

Differential counts are given for 18 cases of acute oral infections. This part of the report is followed by a record of the study of 50 cases from a psychopathic institute. Of these one was edentulous and in three the teeth were not examined. In the remaining 46 cases Rygge found 34 (73-9%) showing chronic periodontal and periapical infections. The diagnosis was for the most part made without the aid of roentgenograms. Blood counts were of 49 of the 50 cases. Ten (i. e. 20.45%) showed a displacement to the left. (Arneth) One of these 49 was edentulous and her blood picture showed this displacement to the left. Rygge was unable to prove that the displacement to the left in the other nine was caused by oral infection. In three cases all the teeth were extracted and six months later the displacement to the left was still present. The results of Rygge's blood studies on patients with periodontal and periapical infections are in harmony with those of Heidemann's on patients with aural infections. In the acute otitides a left-hand displacement occurs; while in simple chronic otitides no (or almost no) change in the blood picture occurs. Acute exacerbations may however, produce marked changes.

Rygge found no blood cell which could be identified with that which Toren (D. Cosmos, Sep. 1922. p. 917) held pathognomonic for oral infection. —*Copy*

12—Schmid, Huldreich. **Untorsuchungen an farbigen Zahnbelägen,** in: Dtsche Mschr. f. Z., Heft 17, 1928. Abs. Ztschr. f. Stomat. xxvi: p. 1129.

These colored deposits consist of bacteria and varying masses of Ca salts. Certain Gram-positive rod-shaped bacteria which require further investigation appear to play the chief role. By microscopical study alone, it is not possible to determine their exact composition. —*Transl. of abs.*

13—Simonton, F. V., D. D. S. **The most significant findings of the California stomatologic research group in the study of pyorrhea,** in: Jour. Dent. Research, viii, p. 235. 1928.

This is an outline of the work undertaken by the California research group in such subjects as mineral metabolism, biochemistry, nutrition, protozoology, bacteriology, etc. It also deals at some length with the question of reattachment following subgingival curettage, based on examinations made by the research group. No formal statement is made on this point as it is thought best to postpone this until after further investigation. The author, however, expresses it as his personal belief "that in certain cases reattachment will take place through the epithelium which covers the treated crevicular surface uniting with the cementum along an *Epithelansatz*." The type of case which appears most promising is one with vertical lesions and marked vascular reaction. The author concludes this part of the subject with the statement "that instrumental treatment of pyorrhea is beneficial; that some regeneration takes place; that the histologic picture of the treated tissue is essentially constructive while that of the diseased tissue is essentially destructive, and that the inflammation in the former is markedly reduced."

In studies on inorganic salt metabolism in dogs the interesting observation is made "that the seat of the disturbance in faulty mineral metabolism is in the tissues rather than in the gastrointestinal tract," and also that calcification is dependent upon two factors; the metabolic state which is inherent in the individual, and secondly, upon diet, since it plays such an important part in regulating all metabolism. "It is believed that the precipitation of bone salts is determined by the balance of ions between the bone-forming cells and their surrounding medium, and that any agent (diet, hygiene, ultraviolet light, active principle in cod liver oil, etc.) which either directly or by catalysis influences the metabolism of the bone cells will alter that balance and thus become an important factor in regulating the deposition and resorption of bone."

—*A. H. M.*

14—Sippy, Burne O., MS., DDS. **Regeneration of bone, peridental membrane and cementum, following experimental injuries in dogs,** in: Jnl. D. Res.—viii. Abs. iii, Feb., 1928. D. Cosmos, August, 1927, in part.

When the injury is confined to the bone, peridental membrane, cementum and dentine and does not involve the pulp, the repair of the bone and peridental membrane is complete. These regenerated tissues are like the old tissues in all essential respects. A marked stage of resorption of the margins of the in-

juries in the cementum and dentine occurs before any regeneration takes place, and then the lost structures are replaced entirely by cementum of the osteoid type with enclosed cells (corpuscles) and typically embedded fibers of a new peridental membrane. Repair takes place alike in teeth with untouched pulps and in teeth that have been rendered pulpless and the pulp canals filled with gutta percha.

—*Copy.*

15—Stein, Gustav. **Plantationsstudien,** in: Ztschr. f. Stomat. xxvi (3), 284-309, 1928. Author's abstr. in English.

In conclusion we may say the following: With all the ways in which we have treated grafts, fusion has followed at least occasionally. When physiological salt solution is employed, glycerine, hypertonic salt solution, etc., fusion follows in about equal proportion. Fusion seems to follow not so easily if Pregl's solution or distilled water was employed, while the use of pieces of decalcified bone is almost always followed by success. In the last case, however, a peculiar process of healing and transformation is followed. If no connection with the bone is effected, the graft may persist very long in the connective tissue in a non-reactive state, without leading to any extended resorption. Deposition of new formed bone only was found when the graft comes into connection with the tissue proliferations arising from the periosteum or marrow space. If no bony junction follows, a greater or less degree of resorption of the living bone nearly always takes place where bone and graft meet. The graft which induces this resorption is not itself affected in anything like the same degree.

Wherever resorption penetrates into a decalcified graft, recalcification precedes resorption. In the non-recalcified parts of the graft resorption lacunas are found only exceptionally.

These findings are thus explained, that the quality of a hard substance influences the process of resorption in such a manner, that some hard-substances are easier to be resorbed than others.

Not only formation of bone, but also resorption seems to depend on a particular differentiation of the germ tissue.

As a practical consequence of the observations with decalcified bone, the surface of the root of teeth, which should be implanted was decalcified in a small degree. Also at the apicotomie the sectional area of the root is decalcified.

—*Copy.*

1929

16—Becks, Hermann, M. D., D. D. S. **General aspects in pyorrhea research.** Pac. Dent. Gazette, xxxvii, May, 1929.

Recent years of increased research have resulted in nearly 350 theories of pyorrhea and an excessive nomenclature which has confused the clinical picture for the practitioner. The term "pyorrhea" (pus discharge) is in no way satisfactory. There are two different disease pictures, namely, "paradentitis," which is characterized by inflammatory processes, and "paradentosis," a disease of the "paradentium" in which degenerative or retrogressive processes are foremost in the pathological activities and which are concealed by paradentitis in the later stage. Weski includes all locally and functionally related tissue complexes: gum, peridental membrane, alveolar process and cementum, under the term "paradentium." In paradentitis there occurs, as a result of inflammation of the gum margin or a heavy layer of calculus, also through toxic irritants at the neck of the tooth, an accelerated detachment of the epithelium from the surface of the tooth. Pathological deepening of the pocket occurs, which is favorable for the deposition of bacteria and food remnants and finally the formation of subgingival tartar occurs. This irritation incites inflammatory processes and the small cell infiltration of the submucosa of the pocket increases. Granulation tissue forms and pus is demonstrable in the pocket. Functional atrophy develops and bone resorption occurs. The clinical picture shows an area of very red or livid blue gum and a very slow and increased loosening of the teeth. Gottleib terms this type of paradentitis "filth pyorrhea" since it exists in patients who take little or no care of their mouths.

Four main clinical forms of "paradentosis" are (1) The presenile atrophy of the alveolar process in which the most important factor for diagnosis is the uniform involution of the soft tissues without pocket formation or

inflammation. (2) Over-pressure paradentosis, where persistent pressure on a bone leads to bone atrophy either vertical atrophy or horizontal atrophy. (3) Paradentosis by lack of occlusion. The functional influences exerted on the bone by the teeth are missing and we find in teeth which lack an antagonist that the alveolar compacta resolves into functional atrophy. The teeth grow into space and cause a paradentitis especially at the point of bifurcation of the roots of the denuded teeth. It is therefore correct to supply each tooth with an antagonist, if it shows signs of being forced out of the alveolus. (4) Diffuse atrophy of the alveolar process without local inflammatory irritants. This, according to Gottleib appears after influenza and other infectious and exhaustive diseases. A clinical loosening of the tooth is noticeable due to resorption sinuses in the bone as well as cementum in which a cellular tissue is laid down.

Cases of pyorrhea which are grouped under the heading of "presenile and diffuse atrophy" are probably the result of a disturbance of metabolism. Observations of the basal metabolism of pyorrhea patients may provide us with material as a key to the solution. Further investigation will fall into three divisions as follows: 1—A purely medical examination by a physician. 2—A thorough dental examination. 3—A biochemical investigation of the blood and saliva.

—*M. M. B.*

17—Beckwith, T. D., Ph. D. and Adrienne Williams, M. A. **Materies alba as toxic material,** in: The Amer. Dent. Surg., xlix, Feb., 1929.

A study of the decomposing white colloidal organic material in the gingival sulcus was made on a patient who had large amounts forming on her teeth, on the assumption of Hatton, Merritt and others that it might be toxic. The authors state that this material may be endogenous and not only from food debris. Microscopic examination showed large numbers of spiral rods and cocci together with quantities of unorganized particles. The material was suspended evenly in water and heated to 60°C for 30 minutes. Half was then reheated for 10 min. in boiling water. Intracutaneous injections of .05 cc. were then made in the arm, using portions of each half. Both caused in six hours a reddened area two inches in diameter and swelling of arm. In 24 hrs. the reddened area was circumscribed to a diameter of ¾ inch and swelling had subsided. After this the two wounds varied; that from the unboiled half in 48 hrs. caused a small vesicle at the point of inoculation which developed into a small area of necrosis surrounded by a narrow red margin which did not heal completely for 21 days. The wound from the portion heated to boiling disappeared in 96 hrs. A wound made by injection of physiological saline was entirely negative. Intracutaneous injections into a rabbit gave similar results; .05 cc. of the portion heated to boiling and then alternately chilled in liquid air and heated to boiling to break bacterial membranes, injected into the patient's skin produced a similar lesion to the boiled portion in the first subject. 1 cc. of the suspension injected intravenously into rabbits led to death in 20 hrs. Cause of death appeared to be intoxication. Blood cultures negative, the only organ affected was the spleen which was enlarged.

The experiments show the materies alba on teeth to be toxic even with the bacteria killed. Since other experimenters have shown that colloidal material (India ink) may be absorbed through the gingival crevice of the guinea-pig, the possible significance of these findings is evident.

—*H. J. L.*

18—Chiuminatto. Luisa. **L'esame del ricambio nelle paradentosi, (Investigation of metabolism in paradentoses,)** in: La Stomatologia xxvii: 269-273. 1929, abs. D. M. f. Z. xlviii (4) 417. 1930.

The expenditure of energy is least during entire rest and hunger. In correspondence with this the metabolism is also at its lowest-basal metabolism. With increase of nutriment the metabolism is also increased and the volume of increase is the "specific dynamic reaction (activity)."

The determination of the basal metabolism is made by means of a Kropf spirometer; the relation of the O intake to the expired CO_2 being calculated at the same time. If this respiratory quotient is about 0.8 normally, its caloric value is about 4.9. (For this daily calculation, see the article.) The

value is less than the normal figure in diabetes, the uric acid diathesis, or endocrine imbalances; in the latter case, it may be increased.

In paradentosis (80 to 90% of the cases) the metabolism is lessened as in all diffuse atrophy. The specific dynamic activity is, however, not lessened in the so-called filth-pyorrhea. Thus, an investigation of the metabolism is indicated to confirm the diagnosis in paradentosis. A lessened spec. dynam. activity is a sign of local atrophy. In such cases the local introduction of O into the periodontal pockets (method of Dunlop) may be useful since it promotes oxidation thus raising the spec. dyn. reaction.

Transl. from Ger. abs. of Ital. Art.

—H. A. R.

19—Oppenot, Mlle. Théréze. ***Contribution à l'étude du pH salivaire.** (Thèse de Paris, 1929) Abs. Rev. de Stomat. xxxi (11) 848-849. 1929.

A fine series of researches which if not directly applicable to therapeutic needs are none the less of great interest. It is an important research in biological chemistry. The first part is a general review of pH with definitions of acidity and alkalinity; and a description of the author's method and technique. Observations were made on:—

1. patients with a stomatologic affection only
2. patients in general medicine together with buccodental symptoms (TB, diabetes, endocrine dystrophies, goitres, etc.)

Conclusions:

a. Gingivo-stomatitis, like tartar renders the pH more alkaline.

b. Buccal suppuration provokes acidosis.

c. Salivary lithiasis appears to entail alkalinity.

d. Non-febrile TB processes alkalinize the saliva; when febrile, they cause acidity.

e. Diabetes, at least in the course of insulin treatment, increases the pH, consequently the salivary alkalinity.

Finally, the author has studied the effects of pH variations on the reducing power of ptyaline. The activity of this ferment is at its maximum with pH normal; lessened with increase of alkalinity or acidity. This diminution which entails an excess of unreduced carbohydrate might favor the development of caries.

(Transl. from French abs.—H. A. R.)

20—Polevitzky, K. M. D. **Blood types in pyorrhea alveolaris,** in: J. Dent. Res., ix: 285, June, 1929.

This study was undertaken to learn whether the distribution of blood types affords any evidence for or against the inheritance of a predisposition to pyorrhea alveolaris. Blood types are known to be inherited . There is considerable evidence that their inheritance is correlated with certain pathological states. Weber and Pastern found a slight increase in group A (N. R. C. classification) among pyorrhea cases. . . . Their findings based on 369 cases were in general confirmed by an examination of 100 cases, the results of which are summarized as follows:

Group	Normal %	pyorrhea %
O	42-45	41
A	40-41.4	49
B	10-10.4	6
AB	5- 5.4	4

—Copy.

MEMORANDA